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Part I.

Original Contributions.

Ophthalmic Miscellanies.

By Wm. Bowman, F.R.S.

No. 1.—*Cases of Malformed, Misplaced, and Dislocated Lenses, in some of which Glaucomatous Symptoms were developed.*

[*Abbreviations used in this paper.*—J = Jaeger's text types. OS = Ophthalmoscope. od = optic disc. F = visual field. T = intraocular tension, as follows: Tn = tension normal. T1 ? doubtful increase of tension. T1 = slight but positive increase. T2 = a higher degree, but the globe still impressible by the fingers. T3 = the eye not impressible, stony hard. − T1 ? = doubtful decrease of tension. − T1 = slight but positive decrease of tension. − T2 = another degree of slackness. − T3 = the globe so soft as to allow the fingers to sink in it. These two last degrees are indefinite. (See "British Medical Journal, October 11th, 1862.) ′ = English foot, ″ = English inch. Hm = manifest hypermetropia (see Donders, on "Anomalies of Accommodation and Refraction of the Eye"), where many other useful abbreviations or signs are given). With the better definition of scientific ideas, such abbreviations become more precise in their meaning, while the constantly augmenting volume of scientific literature makes them more and more necessary. It might be well if the ophthalmologists of all countries, to be represented at the Vienna congress next year, would take steps for some common understanding on this subject.]

The following case is interesting in two respects:—First, as an example of a somewhat rare malposition and

defect of the lens, originating during the developemental period, and illustrating the serious risks to which such imperfect eyes are liable; secondly, in its bearing on the explanation of those morbid variations of intraocular tension, whether by excess or deficiency, which at present form one of the most important subjects of ophthalmological inquiry.

Case I.—The patient was a well-built cabman, æt. 22, who had always been considered "short-sighted." With the right eye at 4″ he could read J1, provided he held the head downward and a little to the right side; with the left eye only J6, and that with the head erect. Both eyes were of good size and appearance; the anterior chambers were of ordinary depth; but the irides trembled when the eyes moved, especially in certain positions of the head; and it was noticeable that when the face was inclined downwards the iris advanced, especially the right, and the nasal side of it more than the temporal. The pupils acted promptly and freely, and when they were expanded the lenses could be seen by the OS to be rather small, and placed too high, as well as somewhat inwards, so that their lower margins crossed the pupil obliquely. The fundus of the eye was clearly seen through the lens in the higher part of the pupil, while through the lower part of the pupil it was seen as in cases of aphakia. Very faint marginal opacities existed in both lenses, only discoverable on close inspection. The man had never experienced any trouble with his eyes till the winter of 1863-4, when he was 22 years of age, and when the following circumstances occurred in connection with the *right* eye, the left remaining quiet.

On three occasions, at intervals of about a week, he awoke with the eye dim and misty, soon accompanied by pain in the eye or brow, but towards the middle of the day the symptoms remitted, and he saw as before, forgetting the occurrence till again reminded of it the following week. He came to Moorfields on the third of these occasions, when, *at* 11 *a.m.*, the tension of the eye was found augmented, T1, there was hardly any anterior chamber, the iris was not tremulous, being almost in contact with the cornea, especially at the inner side, where it was evidently bulged forward by the lens. The pupil was dilated, irregularly oval, and inactive; the conjunctival and sclerotic vessels were

congested, with considerable pain and aching of the globe. The visual field, F, was complete, but vision everywhere dull. Fingers could be counted at 1′. The eye was kept under observation. *At* 11.30 *a.m.* the anterior chamber was found less shallow, the iris not so advanced, the pupil circular and more active, and the pain nearly gone. Tension still T1. *At* 12 *o'clock* the anterior chamber was restored to its natural size, the iris tremulous like the opposite one, the pupil active, the mistiness less, no pain, and T1?

I ordered the man to come daily to my house that I might more accurately study these remarkable phenomena. The next day, February 3, he came at 11 a.m. There had been no return of dimness. The right eye was Tn; he could see J1 at 4″; the lens and iris were in their usual state, as described in the opening statement.

I now directed him to alter his posture, to see whether the lens would vary its position, as he thought the pain and dimness had first come on after stooping. He was made to sit with his head held down in a stooping posture from 11·10 till 11·35, but no inconvenience ensued. When he leaned forward the pupil only became dilated, and when he leaned back, so that the eye looked upwards towards the light, the pupil contracted. Next the head was held downwards and to the right side for 10 minutes, and again downwards and towards the left side, but with no perceptible result.

He was now told to return immediately whenever the symptoms might recur, and he presented himself on the 7th, *at* 11 *a.m.*, saying that the eye had been dim and painful as before since he awoke. He could only see J14, and count fingers at $3\frac{1}{2}$′, T1. The pupil was oval and dilated, the anterior chamber very shallow and unequal, the globe acted, and the conjunctival and sclerotic vessels were injected. By the O S the lens was beautifully seen dislocated forwards obliquely half through the pupil, the outer and lower margin being in front of the iris; there also seemed a general slight haze of the surface of the lens, with fine marginal striæ, not previously so apparent. The red reflex of the fundus was distinct on the inner side, but the od was not distinguishable. This condition gradually passed off as before, without treatment.

On the 9th the same kind of attack recurred, but he did not return to me till the day following, *the* 10*th*. He then stated that

the pain and dimness, instead of clearing off towards noon, had continued all day, getting worse towards evening, and having been severe all night. When he came *at* 11 *o'clock* on the 10th, the pain was great, with much congestion and watering, a very shallow anterior chamber, the pupil oval and dilated, the iris not tremulous, and slightly discoloured. He could count fingers at 2′. No iridization. I expected to find the tension increased, but it was normal, Tn. At 12.15, the pain continuing, a solution of atropia of 1 grain to the oz. was dropped in several times, at intervals of 5 minutes, and seemed to relieve the pain, though it had no effect on the size of the pupil. To take 1 gr. of opium at bed-time, or earlier if pain returns.

11th. Slept well. No pain since yesterday, but the eye much injected, and the iris discoloured. Though the anterior chamber is again of good size, the pupil remains ovally dilated, now probably from the atropia, since it is tremulous, and apparently not displaced by the lens, which has fallen back to its original place, though tilted. Vision very misty. He only counts fingers. The cornea is a little hazy, the vitreous also a little turbid, and the fundus not visible. The tension of the eye was now found not augmented, not even of normal amount, but diminished, — T1.

12th. Still no pain, but the cornea more hazy, with more vascularity of the globe, and he cannot even count fingers. The tension still less —T2. The iris was fairly visible, tremulous, nearly in its natural plane, the pupil smaller and nearly circular (the atropia having been omitted), and the lens not advanced. The fundus of the eye could not be seen, owing to the general cloudiness, chiefly of the vitreous.

It was now necessary to devise some treatment for our patient, or to abandon the eye to its fate. After dislocation of the lens forwards, the globe had become glaucomatous for a time; the heightened tension had then given place to a morbid slackness, and though the lens no longer pressed upon the iris, the congestion was increasing, and the vision nearly gone. If the symptoms could be supposed likely to yield to any general treatment, the eye would still be liable to a recurrence of them all on the next dislocation of the lens; but I could not say I thought it possible to escape the entire loss of the eye by anything but operation. I therefore determined at all events to remove the original cause

of the mischief by extracting the lens, and in doing this to excise a portion of the iris, without indeed having any very definite idea of what the immediate effect of this operation might be on the intraocular circulation and tension, though I could not but watch the effect in these respects with much interest. Chloroform was immediately given, and the iridectomy made outwards because it was in that direction that the lens had projected through the pupil. On completing the section of the corneal margin a bead of vitreous protruded in such a manner as to show that it was still contained within the hyaloid capsule. After the iridectomy the lens was removed without difficulty by a small scoop, a bead of vitreous, remaining in the wound, being snipped off with scissors.

The lens was found to be altogether *too small and too convex*, very like the lens of a young child. Its equator was not circular, but irregular, in a way to give an indication of the trilinear arrangement of the segments, *i.e.*, it approached in a slight degree to a triangular outline, when seen from the front or back. No suspensory ligament was discoverable, so that it seemed that what remnant of this structure had previously held the lens in its position had been broken through, during the operation, at the capsule of the lens, not at the ciliary body. The capsule was entire and quite transparent, and the body of the lens almost, if not quite, transparent also.

During the 13th, 14th, and 15th there was no pain, and the eye seemed to be recovering, becoming gradually less vascular, and the vision better. The tension remained — T3 or 2. The further progress was steadily towards recovery, and may be most simply stated by a transcript of the notes, as follows :—

16th. — T1. Sclerotic injection chiefly about the wound; cornea much clearer. Sight improving; can distinguish faces. 24th. — T1. Slight injection at the wound; cornea quite transparent; good anterior chamber. Diffuse haze of the vitreous. Fundus not seen. With $\frac{1}{2\frac{1}{2}}$ reads J13. 27th. — T1? Vitreous hazy still; reflex better, but od not seen. March 5th. — T1? Vitreous still hazy, and flocculi visible by the OS; the od indistinctly seen. With $\frac{1}{2}$ reads J8. 12th. Tn. The vitreous is clearer, and shreds are seen on it, moving with the movements of the eye. 19th. Tn. With $\frac{1}{2}$ reads J3. 26th. Tn. Slight films still seen in the vitreous; od plainly seen, its colour rather

resembling that of the neighbouring fundus; arteries small. With $\frac{1}{3}$ reads J20 at 15′, and with $\frac{1}{2}$ J1. April 9th. Tn. No morbid vascularity, except just at the cicatrix. The eye is "quite comfortable," and (with the lens) "the best of the two." With $\frac{1}{2}$ reads J1.

In connection with the above case, so far as its interest lies in the glaucomatous state induced by the dislocation of the lens, the two following of traumatic dislocation with glaucoma, may be here related.

Case II.—A clergyman, æt. 37, who had Hm $\frac{1}{12}$ in each eye, and who had formerly been successfully operated on for internal strabismus, chanced to have the right eye hit by a morsel of wood, which was being chopped. After the immediate pain of the blow had subsided by simple treatment, he seemed to be recovering, and did not apply for aid until the 4th March, 1864, three weeks after the accident. Great pain had suddenly come on four days before, and had lasted ten hours. The tension of the eye was augmented, = T1. F complete. Could only see J18. The cornea and vitreous were rather hazy, but the OS gave a fair reflex, and though the od was not visible, there was no manifest hæmorrhage. The pupil was much dilated, and the lens, though quite behind the iris, was seen to have dropped somewhat downwards and inwards, so that its upper and outer edge crossed the pupil above, leaving an interval through which a portion of the vitreous humour appeared to project into the interior chamber. The capsule of the lens was unruptured, and the lens was transparent, but as the light fell in different directions from the OS, dark striæ (which I call *striæ of refraction*, as distinct from *striæ of opacity*) were seen, indicating some derangement of the marginal fibrous laminæ. Such striæ I have very often seen, and I suppose they must have been seen by others, as a condition that long precedes, in certain cases, the opaque or cataractous degeneration of the lens. Here they were probably due to the mechanical injury.

Under strict rest and antiphlogistic measures, with the use of atropine, some improvement occurred, and on the 10th he could see J14, and with $\frac{1}{12}$, letters of J6. But the tension continued T1.

17th. T1. Ciliary vessels injected; front of sclerotic, red. Eye weak, watering, and intolerant of light. With $\frac{1}{12}$, J8.

21st. T2. With $\frac{1}{12}$, J10. Cornea and fundus, duller; lens more advanced, pushing the iris slopingly forward, so as to render the anterior chamber shallow. The eye was more painful. He had also now, for the first time, iridizations; a candle was surrounded by an orange, yellow, and blue halo, the colours not being bright.

I now determined, as in the preceding case, to attempt relief by operation. Under chloroform, a marginal incision of two lines was made upwards and outwards by a Sichel's knife, and a bead of vitreous, which at once protruded, apparently in the hyaloid tunic, was snipped off. A spoon was now passed behind the lens and its capsule, and the lens, rupturing in the attempt, was withdrawn in two or three portions; but as it was transparent, some fragments were probably left. More rather solid, clear vitreous protruding, in all about five or six minims, was now snipped off by scissors, and the eye closed. No iris was removed, as the instruments failed to take hold of it; the vitreous folding it backwards above and behind the corneal section, as usual in such cases. It will be seen that it would have been better if it could have been excised at the time. After operation, —T2.

22nd. No pain; but some swelling of the lids, Tn. Two leeches were applied.

23rd. More pain; the swelling of the lids extends to the temple, and the leech bites are suppurating. Some chemosis. Goulard lotion.

After this the tumefaction subsided gradually. The tension of the eye was then found normal. On the 26th he could see the hand, but could not count fingers. The next day he could count fingers held close; the following day at 4′. On the 30th, with $\frac{1}{2\frac{1}{2}}$, J15. The centre of the section gaping, and the iris protruding there, was snipped off on the 31st. On April 11th, the eye continued red, but pain had quite ceased. On May 10th, redness nearly gone, but returned on use. Tn, with $\frac{1}{2\frac{1}{2}}$, J8. After this the eye became strong, and the vision still improved when he was last heard of in January, 1865.

Case III.—A little boy, $8\frac{1}{2}$ years old, in the middle of May, 1863, had the right eye hit by a child's blunt arrow. The lens was thrown upwards and outwards in the direction of the ciliary body, the lower and inner half of it remaining visible; very little

inflammation followed, and the pupil being kept dilated with belladonna, the eye seemed to be suffering little from the injury, except the impairment of its sight. I did not see him until the 11th of July. The lens was then in the position indicated, its lower and inner margin crossing the pupil obliquely near its centre, and the pupil below this being hazy, and having a dull reflex by the OS. The pupil was also drawn downwards and inwards (*i.e.*, in the direction contrary to that which the lens had taken), probably from vitreous humour which had been forced forwards through it on that side at the moment of injury. The anterior chamber was rather large; the cornea, slightly hazy; the ciliary vessels somewhat injected. The eye was decidedly too hard, T2. As there was little pain, it was doubtful how long the glaucomatous state had existed. Iridectomy, however, and the extraction of the displaced lens being clearly indicated, both were at once proposed, and performed the same day. The iris was excised over the lens, upwards and outwards, and the greater part of the lens easily removed by a scoop. Some vitreous humour was necessarily lost, which was firm and apparently clear.

After this, there was — T for three days, and afterwards Tn until the 25th, when it rose to T1, with a little renewed redness; the sight again less clear than it had become subsequent to the operation. There was no obvious cause for this new increase of tension, and it was treated by rest, atropine lotion, and seclusion of the eye from light.

27th. Still T1, but less redness, and eye quieter.

Aug. 6th. Tn; no redness or pain.

Remarks on the three foregoing Cases.

In the first, the sudden displacement of the lens and pressure on the iris, so as to force it forwards, and drag upon its ciliary attachment, dilating the pupil, seemed to give rise to the glaucomatous state,—to a redundance of the secretions within the eye, and a heightened tension in consequence. This subsided and recurred several times, in apparent dependence on the same cause. Finally, under a continuance of this displacement, and of the congestion

and consequent effusions, the glaucomatous tension gave place spontaneously to the reverse state, viz., a morbid slackness, which is not usual in idiopathic glaucoma, but which must be regarded as a phenomenon of the same order as the hardness, only in the reverse direction—a disturbance of the balance of secretion and absorption from certain vessels within the eye. If the one condition, according to the views of Professor Donders, be the result of a reflex irritation of the nerves controlling the secretions, the morbid softness may have been a consequence of this irritation having passed over into a state of paralysis of the same nerves. Whatever the explanation, the fact is remarkable, not less than the additional fact that iridectomy, with the removal of the lens as the original exciting cause of the whole, put an end to the whole series of morbid phenomena, and restored permanently the healthy state of the vessels, and the proper balance of the tension.

In the second case also (that of the clergyman) the glaucomatous hardness of the eye seemed to originate in the traumatic displacement of the lens and pressure on the iris. It increased as the iris was more pushed forwards towards the cornea; and it was similarly at once relieved by the operation, though the removal of the lens only, with some vitreous humour, sufficed for the result.

In Case 3, the conditions produced by the injury, as well as the remedy, were of corresponding kind.

In neither of these cases of glaucomatous disease, produced by a mechanical cause, was there any tendency to suppuration of the globe, or even to hypopyon, *i.e.*, effusion of pus or lymph into the anterior chamber. In this they correspond with ordinary glaucomatous affections, where effusions are usually serous, rather than fibrinous or purulent. And this has, no doubt, a close and intimate relation with the nature of the morbid process. In iritis, attended by effusion of serum or lymph, and in ordinary hypopyon, augmented tension of the globe is, as a rule, absent. Thus the *type* of glaucomatous disease is different

from that of inflammation. It is essentially an altered balance of secretion, in which intra-ocular secretion, even if of only the natural fluids of the eye, is in excess of the normal quantity, and removal by the vessels does not keep pace with the effusions from them. When pus or lymph are poured out into the chambers or tissues of the eye, as a consequence of inflammation, not of the glaucomatous type, the normal fluids, especially the water of the aqueous or vitreous humours, are absorbed in a corresponding or even an excessive measure, and the tension of the whole organ remains normal, or may be even slackened. Those who would advance at all towards an understanding of the nature of glaucomatous disease, must acknowledge these facts, for they are fundamental. It seems to be a reasonable corollary from them, that glaucoma is, in its essence, not an inflammation; and when inflammatory, only so, as it were, by accident or complication. It is easy to see how serious such an inflammatory complication must become when affecting the vessels of structures confined by their very anatomical arrangement within the corneo-sclerotic case, the limits of whose normal extensibility have been already reached under the distending influence of excessive production of secreted contents, and which must then necessarily re-act on the contained tissues with a baneful pressure proportioned to its suddenness and intensity. The acute and penetrating genius of Von Graefe has sufficiently shown how this pressure must be most peculiarly exerted on the optic disc, and on the circulation and innervation of the retina.

Leaving the subject of glaucoma, I may add to the details of the first related case, notices of a few others in which a similar defect of the lenses was observed, as they may go to furnish materials for a general account of such faults of development.

Case IV.—Aug. 17th, 1858. A little boy of 5 years old, had always been noticed to be "short-sighted," but two days ago a peculiar appearance was noticed in the left eye, and he was

brought to me accordingly. His parents stated that eighteen months ago he had fallen out of bed on his head, that he had been stunned for five minutes, and been convulsed; moreover, that a year ago, he had run his head against a door. These facts had been recollected when attention had been now called to the eye, but I could not suppose them material. I found that both lenses were lower than natural, so that when the pupils were moderately dilated, their upper edge crossed a little below the upper margin of the pupil, the right being the lower. "The pupils act well; and whenever the head is held forwards, the left lens falls partially forwards through the pupil and keeps it distended. Even when the head is not in this position both irides are bulged below by the lenses, and they are very tremulous. There is no pain or inflammation."

I took accurate notes of the above condition, and saw the little fellow from time to time. There was occasionally some injection and watering of the eye; and as the extreme mobility of the loose lenses continued, I was apprehensive lest they might not at some time become detached and fall back into the vitreous humour, or at least become dislocated more completely forwards, and occasion serious inflammation, and even loss of the eye, of which I had seen several examples. Accordingly I extracted each lens by a small upper section, under chloroform. They were helped out clear in their capsule, by a spoon which I had had made for the purpose, a little vitreous being lost. Recovery was in each case immediate. No iris prolapsed, and none was excised; but afterwards, the pupils, though quite clear, were drawn upwards as though after iridectomy, the irides remaining as tremulous as before.

With so young a subject, and an unusually undeveloped mind, it was not possible to ascertain the amount of vision. For objects and colours, however, it was manifestly good.

In April, 1862, he could see J16 at 2″, and was little helped by glasses. Both Tn. The OS revealed no evidence of disease about the yellow spot, but in the right eye there were doubtful signs of œdema of the retina.

1863. Mind that of a child much younger. Cannot give his attention to anything. Memory bad. Head large. Doubtful whether glasses of $\frac{1}{6}$ help a little. To try such.

1865. Intelligence growing. Sight certainly not worse; sees

objects very fairly, so that a stranger would not at once observe any defect of sight.

In the preceding case the want of cerebral development, and the probable feebleness of the retinæ, are interesting in connection with this failure in the development of the lenses. Congenital cataracts may occur in the same relation.

The following case illustrates how useful such eyes may be, provided the retina be sound and the lenses stationary, not swinging about with the movements of the head or globe.

Case V.—T. S., æt. 18, school teacher. Sight as now from childhood; had severe fits, with contraction of hands and feet up to two years old; health tolerably good since, but was thought to be consumptive when 12 years old. The head and face are unusually formed, with large overhanging brows and broad alæ nasi. No peculiarity of the teeth; joints of the fingers large. He has read a good deal without difficulty, as indeed the nature of the employment which he now follows shows, for he has only applied on account of being "short-sighted," thinking that glasses will relieve him. He has several brothers and sisters, all with good sight.

He holds the book very near the eyes, and reads with one eye, the left having slight external strabismus. The pupils are rather dilated and sluggish, the right the more so. F normal. Tn.

The right eye could see Snellen 200 at 4′; with $\frac{1}{8}$ 200 at 10′, without a glass J16 at $2\frac{1}{2}''$, after atropia J10.

The left eye, Snellen 100 at 10′; with $\frac{1}{5}$, 100 at 20′, without a glass J1 at $2\frac{1}{2}''$. Convex glasses helped but slightly.

With the OS the lenses are seen in about the upper two-thirds of the pupils, being displaced upwards, as well as too small and false in shape. For the lower margin, instead of being of a regular curve, presents several sinuosities, and takes on the whole an horizontal direction, as though the lowest part had sprung upwards, released from its attachment to the ciliary body, while the lateral parts were still held by the suspensory ligament. It is remarkable, however, that although the lower, and of course the marginal, part of the lens is thus irregular, the nucleus seen in quite the upper part of the pupil, is perfectly circular, and therefore unconformable in its outline to the marginal part. Its outline is distinguishable, because it is not, like the margin, quite trans-

parent, but faintly opaque; in fact, cataractous, though probably only in a very thin lamina of its superficies; and through it, as well as through the clearer margin, as also below, where there is no lens, the od is easily distinguishable. In both eyes there is the myopic arc, in the right below, in the left outwards.

Remarks.—In this interesting case the lenses were not mobile, being held by the suspensory ligament in rather more than a half of their circumference, and the irides were also not tremulous. Neither was there the risk of dislocation, which attends more extensive separation of the suspensory ligament. From the integrity of shape and perfectly circular outline of the nucleus, compared with the irregular margin of the superficial layers of the lens in the lower part, it may be inferred that the partial separation from the ciliary body took place after the formation of the nucleus, though this was slightly opaque, but before that of the superficial layers, though, as in juvenile cataracts, these were transparent.

The following case, imperfectly noted, was observed in 1855:—

Case VI.—A young gentleman, æt. 15. Very "short-sighted." When he reads, the paper touches his nose. Pupils both active and clear. Irides dark brown, both very loose and floating. Concave glasses diminish objects, and do not enable him to read further off, or to see better at a distance. The pupils do not alter in size when he looks in the same light from a near to a distant object, or *vice versâ*. His sight at a distance is such that he can count fingers at 30 feet.

The eyes are strong for work, and never tire. He never sees false colours or muscæ. He sees alike in strong light and in dusk. The *right* eye has the worst sight, and cannot read ordinary type.

The right eye was examined under atropia. The lens was seen perfectly clear, but rather small, and displaced downwards and outwards, so that its upper edge crossed the dilated pupil obliquely, about one-third from the top. The precise displacement of the left lens was not recorded.

The following cases exhibit a marked family tendency to imperfection of the eyes, of which one example at least is small and displaced lenses, ending in absolute glaucoma.

Case VII.—Mrs. M., between 60 and 70, has been totally blind in one eye 20 years, in the other 15 years, both probably from acute glaucoma, according to the history.

Case VIII.—Mr. M., æt. 43 (son of Case VII.). Has always had imperfect sight. Both lenses have delicately-traced opacities on the front, those of the right being the fainter, those of the left the denser, but with clear interspaces. As his sight was stationary for many years, these opacities have probably existed from an early age. Lately he thinks the sight has been worse. He uses the right eye for reading, and the left for distant objects, using a concave glass of $-\frac{1}{20}$. Lately the eyes redden on use. There is insufficiency of the int. recti on looking at near objects, the left eye then moving outwards. As he managed to pursue his avocations no operation was recommended, though such may become necessary. Being found to read more comfortably with a convex glass of $\frac{1}{30}$ and prisms of 3°, these were ordered, with an eye-douche.

Case IX.—Mrs. C., æt. 38 (daughter of Case VII.). Sight always imperfect, and has used atropia for many years. Without atropia J18; with atropia J7 and 8. Both eyes present good examples of juvenile nuclear cataracts, the superficial layers of the lenses being clear. She has always read with comfort, but applies now because the atropia has lately irritated her. Another form was ordered, with an intimation that an operation for displacing the pupil would be applicable in case of the belladonna still being unmanageable. For three years I have not heard of her, so that I conclude it succeeds.

Case X.—Miss M., æt. 33 (daughter of Case VII.). Absolute glaucoma of both eyes. Both T1. The right has a singular abrupt and prominent ciliary staphyloma upwards and inwards, the size of half a pea. She was "very short-sighted" as a girl, but could see small objects well with the right eye if they were held very near to it. The right gradually failed 12 years ago; the left also gradually six years ago. No perception of light for three years.

The pupils are widely dilated. Both lenses are clear, and are

displaced upwards and inwards so much that only one-fourth of each is visible in the large pupil. The fundus of the eyes lights up well, there is slight glaucomatous cup in each, and the retinal vessels are atrophied. The loss of sight seems to have been due to simple glaucoma of the ordinary kind.

Remarks on the last four Cases.—They illustrate a fact I have often noticed, viz., that a family tendency to defect or disease in the complex organs of sight, though often manifesting itself in even the exactest identity of form, yet may do so in other cases with variations, as here: *a.*, displaced but clear lenses; *b.*, rather peculiar form of early cataract; *c.*, the more common form of nuclear juvenile cataract; *d.*, glaucomatous affection;—the latter being the most subtle, and referrible perhaps to some inherited disposition of the vessels or nerves, which may be supposed to be the primary seat of that disease. It may, however, be added that I have noticed a decided tendency to glaucomatous disease in persons who have undergone, *late in life*, an operation for juvenile cataract—the same operation which at an earlier period would have been perfectly well borne, inducing in them an intractable glaucomatous reaction, and constituting one of several valid grounds for not too long delaying operative relief. I have attributed this to the long strain to which such eyes have been exposed in looking always through the dull medium—a strain alike of the retina, of the ciliary apparatus, and of the external muscles, and manifested often by redness, watering, and distress. But yet glaucoma, independently of this, is often decidedly hereditary, or a family complaint.

CASES OF NEURO-RETINITIS ASSOCIATED WITH KIDNEY DISEASE.

By J. W. Hulke.

Assistant-Surgeon to the Hospital, and to the Middlesex Hospital.

The association of impaired sight with kidney disease was recognised as far back as 1839 by Addison, and an article was written upon it by Landouzy, in the *Gazette Médicale de Paris*, ten years later; but our knowledge of the tissue-changes, on which the visual defect depends, and of their objective signs, is of recent date; it belongs to the era of the ophthalmoscope, and has been chiefly worked out by our indefatigable German confrères.

The ocular affection, erroneously regarded by Landouzy as the precursor of the renal, usually first appears when the kidneys have become granular and contracted, and when the left ventricle of the heart is hypertrophied.

The visual defect assumes two forms, the one a rapid diminution of acuity, occurring simultaneously with other disturbances of the cerebro-spinal system; as drowsiness, convulsions, delirium; the other a slowly progressive diminution of acuity, interrupted by occasional accessions of greater dimness, with contraction and mutilation of the visual field, not ending in absolute blindness, and susceptible of improvement.

In connection with the last form, these morbid changes are found in the optic nerve, retina, choroid, and vitreous humour. The optic nerve is swollen by the presence of an excess of extravascular serum, it is œdematous, and by the proliferation of the corpuscles of its interstitial connective tissue. The ophthalmoscopic effects of this are that the outlines of the tumid nerve are hazy or hidden, the normal reflexion from the lamina cribrosa is absent, the central vessels are indistinct and cannot be traced

backwards from the surface of the nerve. Around the nerve is a zone of grey œdematous retina in the inner layers of which very singular bodies occur which have given rise to much discussion.

They bear some resemblance to ganglion cells, and were at first supposed to be such, enlarged and sclerosed. In vertical sections, tangental to the optic nerve-entrance, they are found scattered or clustered in nests, packed in the arcades formed by the inner ends of Müller's connective radial fibres. Where the clusters are large, they slightly project the inner surface of the retina, and encroach upon and displace the layers at their outer surface. They have been observed to reach nearly as far outwards as the layer of Outer granules, and at this stage it would scarcely be possible to say in which layer they originated.

Their ganglionic origin was shortly disproved by the observation, that at an early period, when they are scattered, or when the clusters are still small, they occur in the layer of optic nerve fibres; this led to the conjecture that they were peculiar transformations of these fibres, and the subsequent discovery of their apparent continuity with and passage into the nerve fibres appeared to finally put their source beyond doubt. Besides these nodal thickenings, a uniform swelling of the nerve fibres, attributed to œdema, has been described by some.

Two alterations are observed in the connective tissue elements, hypertrophy with sclerosis, which chiefly affects the stout inner portions of Müller's radial fibres, and the interstitial tissue (the glioma after Virchow's nomenclature) derived from these; and fatty degeneration, which principally affects the fine terminal branches of these fibres in the outer layers.

This disintegration of the connective tissue has been regarded by some as the source of the large granular corpuscles which occur in great numbers, principally in the inter granule and outer granule layers, the opinion resting

chiefly on the visual improvement which occasionally follows the disappearance of the white dots which they form when clustered. These dots run together in a wall around the grey zone encircling the optic nerve, they also stipple this opaque zone, radiate in lines from the fovea, and form small glistening white islands in the transparent retina. The fatty metamorphosis of the glioma may be one source, and, perhaps, the disintegration of extravasated blood is a second, but I think it not improbable that the fatty transformation of the excessive hyper-albuminous and fibrinogenous serum constituting the œdema is the principal source. The structureless and fibrillated coagula which form such conspicuous objects in vertical sections are evidently referable to this exhalation. The structureless coagula also principally occur in the inter-granule layer. They range from the size of a blood disc to large irregular nodules and are often branched. They have a peculiar pellucid aspect, some acquire a very deep tint from carmine, while others remain almost colourless after long immersion in the staining fluid, and sometimes different parts of the same nodule will be differently affected by the dye.

In describing the large granular corpuscles, I referred to extravasated blood discs. These escape from ruptured capillaries. I do not recollect in this class of cases one instance of hæmorrhage from the larger vessels. The capillaries are dilated, and sacculated and attenuated by the increased pressure on their inner surface, which results from the disturbance of the normal attraction between the circulating blood and the tissues arising from the presence of urinary constituents in it, from the compression of the veins in the swollen optic nerve, and from the increased impulsion of the blood-column of the hypertrophied ventricle; or the walls of the capillaries are thickened by an amyloid deposit, and their calibre proportionately narrowed. Amyloid thickening of the arteries has been anatomically verified, and I have found the muscular coat

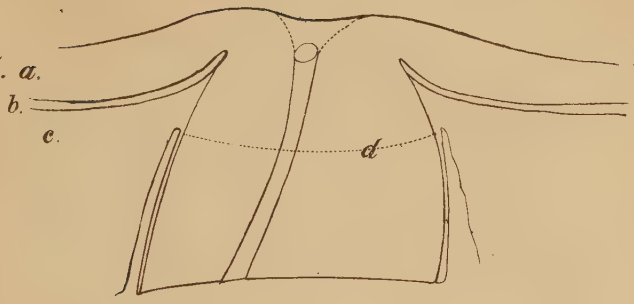
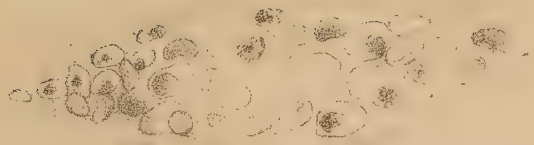
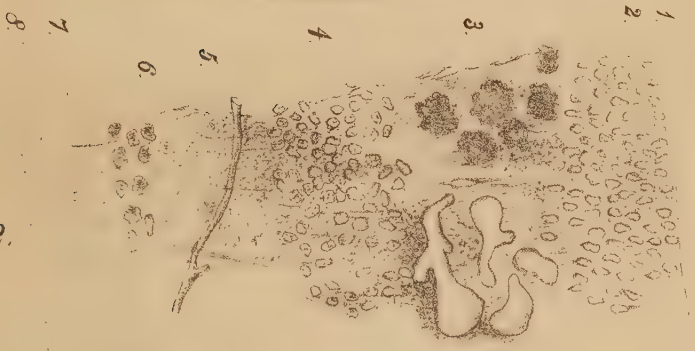


Fig. 4.





EXPLANATION OF PLATE.

(*To face page* 18.)

FIG. 1.—A vertical section of the optic nerve through its centre. *a*. Retina. *b*. Choroid. *c*. Sclerotic. *d*. Posterior limit of the lamina cribrosa.

FIG. 2.—A vertical section of the retina near the optic nerve and radial from it. × 240.

1. Membrana limitans externæ. 2. Outer granule layer. 3. Inter-granule layer, containing nodular coagula and large granular corpuscles. 4. Inner granule layer. 5. Granule layer. 6. Ganglionic layer. 7. Optic nerve layer. 8. Membrana limitans interna, with roots of H. Müller's connective radial fibres.

FIG. 3.—A nest of sclerosed nerve-fibres? *in* the optic nerve layer.

1. Normal optic nerve fibres. 2. Sclerosed ditto. 3. Membrana limitans interna. 4. H. Müller's connective radial fibres.

FIG. 4.—Isolated sclerosed nerve-fibres from the above section.

much hypertrophied. The veins are swollen. In the opaque region both the veins and arteries are concealed wherever they dip from the surface, with the appearance of frequent breaks in their continuity. The tissue-changes in the choroid are less known than those in the retina. Amyloid deposits in the chorio-capillaris is one of the most important, since such a condition of the capillary membrane is calculated to hinder osmosis, and to impair the nutrition of the bacillary retinal elements.

The alterations which have been observed in the vitreous humour are a finely granulated haze, proliferation of its corpuscles, and the irruption of blood discs and fibrillated coagula from the ruptured retinal capillaries.

These textural changes in the retina and optic nerve form the "chronic retinitis" of some, the "retinal degeneration of Bright's disease" of other ophthalmic surgeons. While any of their equivalent ophthalmoscopic signs is enough to excite a suspicion of retinal disease, it has been said that in order to possess a pathognomonic value they must be collectively present, and definitely grouped. The swollen nerve should be encircled by an opaque grey retinal zone; this in turn surrounded by a white belt, and the yellow spot marked by radial lines of white dots.

According to my own experience, such a disposition does not occur in the majority of cases. In many cases some signs only are present, and their value in these is lessened by the fact that every one of the textural changes denoted by them has been observed where kidney disease could not be detected, notwithstanding careful and frequent examination, chemical and microscopic, of the urine. Although cases are not unfrequent in our out-patient room I had never been able to procure material for an anatomical investigation until some months ago, when I obtained the eyes of two patients. An account of these, with short notices of some other cases taken from my note-book, may not be uninteresting to our readers.

CASE I.—*Neuro-retinitis—Granular, Contracted Kidney, and Hypertrophy of the Left Ventricle of the Heart.*

February 24, 1864.—A journeyman confectioner, æt. 20, came to the out-patient room complaining of great dimness of sight. He told me that a week before, while at the theatre, his sight became so dim that he could not recognize the actors.

I regret that I have mislaid a note which I made of the state of his vision, but I recollect that its acuity was so diminished that he could not follow his work, which was candying fruit, and he was led into the room.

Both optic nerves were grey and hazy, their outlines were obscured, and within their area the central vessels were indistinct. In the left retina several crimson, brush-like patches of recently extravasated blood radiated from the optic nerve nearly to the equator; and in each eye around the optic nerve there were several small buff spots, which, from their relations to the large venous branches and their shape, I referred to the inner retinal layers. They were, probably, retrograding extravasations. The turgescence of the retinal veins beyond the limit of the optic nerves, where the retina was transparent was very striking.

I found his urine highly albuminous, and learned that for a long time his strength had been declining. He was very thin, pale, and had a troublesome cough.

At his second visit a few days after I found him so weakened by profuse epistaxis that my colleague, Dr. Stewart, took him under his care in the Middlesex Hospital, where he died April 24th of erysipelas.

Dr. Cayley, our demonstrator of morbid anatomy, kindly gave me the following extract from the register:—

"P.M. Reg. No. 1813. Body emaciated. Face very œdematous. Slight general œdema. Lungs œdematous; cretified tubercle in the apex of the right. The pericardium contained ʒiii of cloudy serum. Its serous surface was congested and ecchymosed, and the heart's base was covered with soft yellow lymph. The heart weighed ʒxiv. Its left ventricle was much hypertrophied; the valves were normal. A few small, scattered, atheromatous spots were seen in the ascending aorta. Both kidneys were very small; the right weighed ʒiv, the left ʒiii

only. Their capsules adhered too firmly. Their surfaces were granular, and studded with small cysts. Their texture was tough. The thickness of the cortex varied from 2‴ to 3‴, and its colour was reddish. The gastric mucous membrane was congested, and near the pylorus it was ecchymosed."

The eyes were placed in chromic acid. When hardened I cut the left across at the equator, and carefully removed the vitreous humour from the posterior half. The optic nerve-entrance now appeared unnaturally prominent. In a vertical section laid through the centre the edge of the sclero-choroidal ring was seen to be directed forwards, while the lamina cribrosa was unnaturally concave. Evidently this proceeded from swelling of the intra ocular end of the nerve. The swelling was produced by an abundance of small cells and nuclei, derived from the corpuscles of the glioma. They were imbedded in a granular substance, and formed dark bands between the transparent bundles of nerve fibres. The inner surface of the retina around the nerve glistened, owing to the presence of minute shining dots, clustered in small patches, and streaks which radiated from the nerve in all directions, except at its temporal side, where they formed lines concentric with it across a fold which ran from the fovea towards the nerve. These minute shining dots were strictly confined to the retina. None occurred within the area of the nerve-entrance, their first appearance beginning at the distance of its diameter from it. The glistening region was also marked by a few dull, grey, slightly raised spots, ranging from a fine dot to $\frac{1}{3}$‴; and the retina nearer the equator, which did not glisten, exhibited a few scattered, dark, brown-red extravasations of blood.

The entire venous system of the retina was dilated, even the small peripheral veinlets were conspicuous. The trunks were hidden in the glistening central region wherever they dipped from the surface, and they wholly disappeared from view at the distance of 2‴ from the centre of the nerve owing to the greater turbidity of the tissues here. Vertical sections showed that the retina, particularly around the nerve, was thicker than natural, which in great part proceeded from a loosening and separation of its elementary tissues, such as œdema might have produced. With the exception of the bacillary, the layers were well preserved. The glistening region

contained great numbers of large, compound, granular corpuscles, which were most numerous near the nerve, and became fewer peripherally. They were chiefly restricted to the inter-granule layer, but when very large or in clusters, they encroached upon the inner and outer granule layers.

The inter-granule layer also contained structure nodules, some of which were very large and branched. Most of them were deeply stained by carmine, while others were scarcely tinted by it. They were, probably, albuminous coagula. The muscular coat of the arteries was hypertrophied, and the capillaries were sacculated.

CASE II.—*Neuro-retinitis—Contracted, Granular Kidneys—Hypertrophy of Left Side of Heart—Death—Dissection of the Eyes.*

In October, 1864, a pale, feeble woman, æt. 44, came under my care, at the Moorfields Hospital, for ophthalmia tarsi and styes.

After a long absence she was brought December 24, in consequence of failure of her sight a week before. With the right eye, which began to fail a couple of days before the left, she read words in No. 20, and with the left eye No. 16 of Jaeger's types at 6″ distance.

The optic nerve of the right eye was swollen, opaque grey, and surrounded by a zone of greyish, cloudy retina, dappled with dots and brushes of extravasated blood, which radiated from the nerve towards the equator. The central vessels appeared to end at the surface of the nerve, they could not be followed towards the lamina cribrosa, and their larger branches, though swollen, were occasionally hidden in the zone of opaque retina. The ophthalmoscopic signs in the left retina were like those in the right, but less strongly marked. Her urine was albuminous, and she was so gravely ill that I procured her admission into the Middlesex Hospital, under Dr. Stewart, who has kindly supplied me with the following extract from his clinical clerk's book.

———— admitted January 24, 1864. "A woman of fair muscular development and moderate stoutness, complexion dark, a native of Wiltshire, married 25 years, had nine children. Catamenia ceased three years ago. Never had a serious illness

till the present, which began two years ago with weakness and loss of flesh, followed by œdema of the legs. About a month ago had rigors, after which the sight of the right eye became dim, and the next day that of the left eye failed in the same way. After the rigors she kept her bed from a general feeling of illness. Vomiting then began, it sometimes occurred two or three times a day, but was occasionally absent for a day or two. At the date of her admission into the hospital her face was puffy, and her skin coarse and dry; urine very pale, sp. gr. 1010°, depositing a slight cloud of albumen. Pulse 96, soft and very full, occasionally dichrotous. Diastola rather accentuated at the base; systolic bruit at the left edge of the sternum; respiration calm; slight sibilus on the right side."

"The vomiting persisted obstinately. A fortnight after admission the lungs became congested, but this subsided in two or three days. A week afterwards she had copious epistaxis. February 20, she had a fit which lasted about five minutes, both sides of the body were equally convulsed. It was followed by considerable drowsiness and depression. There was no foaming, and the tongue was not bitten. Next day she had a second fit. After this she was drowsy and her mind was confused. Her breathing was almost stertorous. Epistaxis again occurred, other fits followed, then congestion of the lungs, coma, and death, for twelve hours before which there was total suppression of urine."

Dr. Cayley has given me the accompanying note of the post mortem examination made February 22, 1864.

"The heart weighed 13 oz., the wall of the left ventricle was very thick. The edge of one curtain of the mirtral valve was thickened. The right pleura contained a few ounces of serum. The kidneys were very small, weighing only $1\frac{3}{4}$ and $2\frac{1}{2}$ oz. Their surface was granular. The medullary substance of the supra-renal capsule contained recently extravasated blood."

The left eye was opened at once. Its optic nerve-entrance and the surrounding retina were swollen and opaque; and some small patches of extravasated blood were grouped about the yellow spot. The right eye was immersed in a solution of chromic acid and afterwards examined with particular reference to the minute textural changes in the retina and nerve. These, with one exception, were identical with those described in the preceding case, the

difference being the sparing occurrence of the bodies, now commonly regarded as hypertrophied nerve fibres. These occurred in small clusters, in very few instances in large nests, in the layer of optic nerve fibres. Many contained one, some two nuclei, which had a homogeneous or coarsely granulated aspect, and took a deep red stain from carmine. The other cell contents, or the material surrounding the nucleus, had a lower refractive power than the nucleus, a finely-granulated texture, and were but lightly tinted by the same carmine solution. I failed to trace any connection between these bodies and the nerve fibres among which they were embedded.

CASE III.—*Neuritis, eventuating in Atrophy—Retinal Hæmorrhages —Disseminated Choroiditis—Waxy and Granular Casts.*

A delicate woman, æt. 38, came to the hospital September 16, 1865. She said that she had been confined with her first child in the preceding July, and that a few weeks before this her sight began to get dim, and her legs swelled. Her strength had not returned since her confinement. She had leucorrhœa, water-brash, and albuminuria, and the urine also contained waxy and granular casts.

With the right eye she could read No. 12, and with the left No. 6 of Jaeger's types, at twelve inches distance.

Both optic nerves were dull-grey. Their outlines were confused. The retinal arteries appeared to be contracted, while the veins were irregularly swollen. Towards the equator the choroid was thickly sprinkled with minute black dots. They were sharply defined, and the details of the surrounding structures were distinctly visible through the transparent retina. I prescribed tinct. ferri. muriatis, apparently with benefit to the renal disease, since she gained flesh and strength, but her visual acuity continued to diminish, so that October 21st the left eye read No. 16, and the right No. 12, Jaeger's types.

December 23rd.—A still further decrease was noted, the left eye reading No. 18, the right No. 16. The turgescence of the retinal veins was less. The black choroidal dots were numerous. Some of them were fringed with a very narrow, whitish ring, in which the disappearance of the choroidal epithelium exposed the

sclerotic to view. In the left eye the black dots had invaded the area of the yellow spot.

May 27, 1864.—A still larger number of the black choroid dots were seen in each eye. The optic nerves were anæmic, and the details of the lamina cribrosa were unnaturally distinct up to the very edge of the nerve which was slightly blueish. In the right eye, upon and below the yellow spot, were four crimson patches of extravasated blood, and a still larger fifth patch below and at the nasal side of the nerve.

February 13th.—Much of the extravasated blood had been removed. The details of the choroid were now dimly perceptible through the largest patch. The visual acuity of the right eye had slightly increased, and that of the left in a very remarkable degree, for this eye now read No. 1 slowly. A fortnight later all traces of the extravasated blood had disappeared. She complained of increasing weakness, and a few days afterwards miscarried of a two months fœtus.

March 17th.—The optic nerves are more anœmic, and the details of the lamina cribrosa are still plainer. There are a few small, scattered extravasations of blood in the right retina.

April 23rd.—The left optic nerve is very white, and the arteries are so contracted that the primary and secondary divisions only are traceable, and this for a short distance. The black dots are still numerous in the choroid near the optic nerve, but have left the equator. They have entirely disappeared from the right retina. The visual acuity of the left eye, which had improved so remarkably that Feb. 13, No. 1 was read, is now diminished, that even No. 20 is not perceived. There is a slight improvement of that of the right eye (No. 12). She says she is stronger than she has been for a long time, and she looks better.

CASE IV.—*Neuritis—Amyloid Thickening of the Arterial System of the Retina—Albuminuria—Apoplexy—Death.*

A painter, æt. 54, happening to close his left eye, was surprised that he did not see with his right. Eight months after this, his left eye becoming dim, he was brought to the hospital, October 18, 1862.

The right eye was still slightly sensitive to light. Its optic

nerve was dull, greyish white; the vertical portions of the central vessels, and the lamina cribrosa were hidden. The retinal arterial tree had a most singular appearance which I had never noticed before. The primary and secondary divisions of the artery in the area of the nerve, and in the retina, resembled stone-white cylinders, marked with an interrupted axial red streak, where the coats were thin enough to allow the blood to be seen.

In some places the red blood streak was very uniform, while in others its breadth was irregular, its edges were pale, and its ends tapered. The lengths of vessel exhibiting the red streak were separated by other lengths entirely white, from which it was wholly absent. In the secondary divisions the red axial streak was proportionately wider than the primary, and occasionally it occupied the whole breadth of the vessel, to the complete interruption of the white border. In the tertiary divisions the interruption of the opaque margin was more frequent, and the finest arterial branches exhibited only a linear series of opaque white dots, between which the vessel seemed to be healthy.

With the left eye he recognized such objects as a finger, but could not spell the largest type. Its optic nerve-entrance was reddish, and opaque. The veins within its limit were hidden, while beyond they were very swollen. The vitreous humour was hazy.

He habitually drank to excess. His complexion was sallow, and his appearance haggard. His urine had a high specific gravity, and was highly albuminous. I saw him once only, and I recently heard from his daughter that he died soon after of apoplexy.

A NOTE ON "INTERMITTENT GLAUCOMA."

By Ernest Hart,

Ophthalmic Surgeon in St. Mary's Hospital, London, and Lecturer on Ophthalmic Surgery in the School.

The following brief details will afford a view of a variety of glaucoma, which, I think, deserves to be distinguished as intermittent glaucoma. The study of cases of this type will probably afford material for a nearer comprehension of the pathology of that disease. They present, at least, an interesting and suggestive phase of the affection.

A lady beyond the middle age, and some years past the critical period, having enjoyed excellent health up to the time of her present attack, applied to me in the summer of 1865 to examine the left eye. She was suffering acute pain in the eyeball. It was deep seated, and extended over the brow. When she saw me she was completely blind with that eye, not having even the capacity of perception of light and darkness. The eyeball was of stony hardness. The pupil was dilated ovally, and immovable; the superficial vessels somewhat, but not greatly, enlarged. She was not much alarmed about it. She told me that her eyes had been perfectly healthy until three weeks previously; that she had then suddenly felt some pain, and slight accessions of dimness of vision, and had consulted an eminent ophthalmic surgeon, whose opinion is justly of great weight, and who had not found at that time any cause for apprehension, but had prescribed a slight general treatment. This had seemed to relieve the uneasiness of the eyeball at the time, but a few days afterwards the right eye became suddenly more dim, then failed altogether, the next day greatly recovered, so that she could read "small print." For the few days before she came to me the pain had been more acute, and the vision had gone. She anticipated, however, a return of sight, probably in a day or two, and was chiefly induced to call on me by the

anxiety of her friends. The ophthalmoscopic signs confirmed entirely the external symptoms, and I felt bound to state to her friends the serious conditions present, and to advise iridectomy. I repeated this recommendation urgently in consultation with Dr. Bradley, of Greenwich, and subsequently in consultation with Mr. Bowman, who supported that recommendation. Professor Donders happening to be in London at this time, Mrs. C. had also the benefit of his opinion, and Professor Donders also strongly urged the same view. A further period of total blindness and acute pain at length reconciled her to accept the proposition, which she held, however, in great horror, and the more so that after a fortnight or more of complete loss of vision it was not possible to make any but a dubious statement of the probable results of iridectomy. The vitreous was now cloudy, and the fundus could not be well illuminated, but there was no loss of polish of the cornea, nor was the superficial congestion much increased. Assisted by Dr. Bradley, Mr. Mahon, of St. Mary's, and Mr. Teevan, of Westminster Hospital, I performed an ample upper iridectomy for her on the 13th July. There were no difficulties in the operation, which was uncomplicated. There was no effusion of blood into the chambers of the eye. The immediate effect of the operation was to relieve all pain. The patient, although of the most highly excitable temperament, and suffering greatly both in mind and body during the two previous days, displayed a remarkable equanimity after the operation. She suffered nothing, she said; and at the end of four days, when I just raised the lid, declared that she could see my features indistinctly. Five weeks afterwards she could read clear print (No. IV, Giraud-Teulon) with ease.

The second case which I will mention is one of a very puzzling character, and in which there were singular complications. The patient was an unmarried lady, under the care of Mr. Noverre, of South Street, Park Lane. She was of highly nervous temperament, and had been subject to a

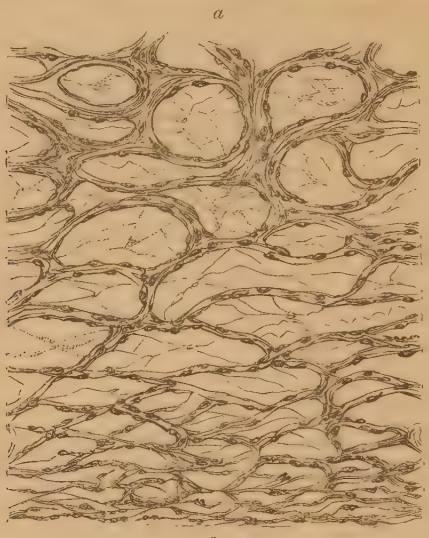
variety of hysterical affections in succession. I was called to her in October, 1864, and learnt the following particulars: She had been suffering during July and August from dubious head symptoms, which were either wholly or in part hysterical. In September she was seriously ill, and confined to bed, unable to have her meals, and for some days apparently insensible. She had been seen by Drs. Williams and Quain, who, I believe, regarded her anomalous and varying symptoms as mainly of hysterical nature. Towards the end of August, however, after a "seizure," in which she had been unconscious, both eyes were much inflamed, and the pupils dilated. She complained that she could not see much, and that she was nearly blind with the right eye. The condition of the right eye seems, however, to have improved gradually, until the sight might be declared nearly natural, but the left grew worse, so that three weeks after, when I saw her with Mr. Noverre, I found the eyeball much inflamed, scarlet injection of the conjunctiva, and turgidity and flexure of the subconjunctival vessels. The pupil fully and immovably dilated. The eyeball was not very tense; in fact, but little more than normal tension was present. The optic disc was, however, cupped, and the veins dilated. The vitreous was not clouded. The pupil of the right eye acted freely, and there was no abnormal ophthalmoscopic appearance visible within the eye. There was, however, considerable presbyopia. I did not ascertain its precise degree, owing to the excitability and distress of the patient: it was described as of quite recent invasion. The sight of the left was gone, not even qualitative perception being left, and the patient declaring herself insensible to the presence of a powerful light in a dark room. Under these circumstances, I considered the case to be one of absolute glaucoma, but having peculiarities in regard to the moderate tension of the eyeball, and the clearness of the media. I counselled iridectomy, but feeling the difficulties of the case, I advised a further opinion, and met Mr. Critchett in consultation

next day. He concurred in the opinion I had expressed, and advised iridectomy without any delay, kindly offering to assist me that day in the operation. The patient, however, could not make up her mind that day, but next day she was desirous of having the operation performed at once. Mr. Critchett having been called from town, I operated with the assistance of Mr. Noverre, Dr. Anstie giving chloroform. While completing the iridectomy, a slight movement occurred of the patient's head, which lead to a partial dialysis of the iris extending from the incised edge. There was some effusion of blood into the anterior chamber. The pain over the brow, its congestion, and irritability were set at rest by the operation, but there was not the slightest improvement in the vision, even after the blood effused had been absorbed. The patient has remained totally incapable of appreciating light from darkness. Having the peculiar characteristics which sometimes exist in highly hysterical women, she has since endeavoured to produce a simulated paralysis of the right pupil by instilling atropia drops in the eye. She had heard the matter of dilatation much discussed during her previous illness, and became aware of the local uses of atropia. The simulation was, however, palpable. The dilatation of the pupil by atropine is, indeed, always discernable by the surgeon who is accustomed to ophthalmic investigations, and does not resemble that of glaucoma, being more complete, and unaccompanied by the characteristic signs. Moreover, prior to operating, I had instilled one drop of the solution of calabar extract (1 drop = 4 grains of the bean), which is insufficient to overcome the dilatation of even feeble atropization, but sufficed to contract firmly this pupil, so as to facilitate its excision.

The very marked and complete intermission of the glaucomatous affection in the first case, its alternation in the second, and the subsequent supervention of absolute glaucoma of the eye, in each case, are clinical circumstances which seem to me worthy of note. Intermissions

so marked are indicative of the nervous relations of the malady. The peculiar temperament of these patients may have tended to bring this element into especial prominence in their cases. The view which supposes that iridectomy acts beneficially by arresting the progress of hypersecretion by the exsection of the nervous plexus and ganglia of the ciliary is that which accords best with the disposition of the parts. The dilatation of the pupils, and the relaxation (tortuosity and engorgement) of the vessels in glaucoma are pathological circumstances which point to the same conclusion. As to the action of iridectomy being beneficial by section of the ciliary muscle, or in reducing its tension in spasm, I am entirely sceptical. The stated circular muscle described in connection with the ciliary has, I am convinced from repeated investigation, no existence as a separate muscle. In the bird there are assuredly no circular muscular fibres whatever in the ciliary region, either in the position assigned to them in the human eye by Muller, or in any part of the muscle. I have very numerous sections and preparations which will sufficiently prove this. In the human eye, in a vertical ciliary section, cut ends may be seen of oblique fibres, which may or may not be muscular; it is difficult to decide whether these fine soft fibres are muscular or fibrous, for the human ciliary altogether is composed of fine soft unstriped fibre. The fibres, however, correspond to connective tissue fibres in birds, where the distinction is, of course, easy enough, as both iris and ciliary of birds are made of striped fibre. However this may be, there is certainly no *circular ciliary muscle* in the human eye, and a section such as that effected in iridectomy could have no effect in reducing the tension of the muscle. In examining the tissues of the eye, I have been very much struck, not only with the mass of nerve fibres which form a plexus amongst the fibres of the ciliary muscle, and the number of ganglion nerve-cells discoverable there, but with the great plexus of nerves which is supplied to the iris, and which are not,

perhaps, so fully known to anatomists, although I see that they have been well described by Donders. As I have not



seen them mentioned in the most recent descriptions or in the anatomical prefixes to Wecker's recent and excellent book, or figured anywhere, and they are of especial interest in connection with iridectomy, I may be permitted to append a drawing of the nerves of the iris of a bird from a preparation which I possess, and shall be happy to show to those interested.

The drawing was very kindly made for me by Mr. Lockhart Clarke, as I have not the necessary skill as a draughtsman; and such drawings are best made by those who have some anatomical knowledge of the object. It represents these nerves as seen under an object-glass of $\frac{1}{7}$th inch. The plexus is made up of small bundles of nerve-fibres at the ciliary margin *a*, but near the pupillary margin these cords become progressively finer, and ultimately are reduced to single nerve-fibres. The iris was prepared in chromic acid solution and then rendered transparent by soaking in glycerine and coloured with carmine.

ON THE EFFECT OF INJURIES TO THE FIFTH NERVE, ON THE NUTRITION OF THE EYE-BALL, AND THE FUNCTION OF SIGHT.

(Continued from Vol. IV, Page 381.)

By Jonathan Hutchinson, F.R.C.S.

In a former paper on this important subject I have recorded cases in which diseases of the trunk of the fifth nerve, occasioning anæsthesia, were followed by destructive inflammation of the eye-ball,* and some of which seemed to me to favour the old belief that this nerve is of direct efficiency as regards the maintenance of the normal nutrition of the eye. Subsequently† I have alluded to other less direct cases, in which reflex disturbance of the nutrition of the eye-ball or of the centres of vision, seemed to have followed injuries to branches of the fifth at a distance, or even persistent irritation of a distant branch, such as that caused by tooth-ache. On this question the doctrines prevalent some years ago were clear and decided, and supported the belief that such reflected consequences were not infrequently observed. That an injury to the frontal nerve, for instance, was capable of causing amaurosis, no one doubted. On this point, however, as well as regards the direct influence of paralysis, modern physiological experimenters have been sceptical. Relying partly upon the circumstance that experiments on animals have been without result, and partly upon the inconclusive character of the clinical facts as yet recorded, a considerable number of our best surgeons at the present day are incredulous on this matter.‡

Such being the state of opinion, it seems very desirable

* See Ophthalmic Hospital Reports, vol. iv, page 189.

† See ibid., page 381.

‡ The list of the heterodox does not include the name of Dr. Brown-Sequard, who is a vigorous supporter of the old opinion as to reflex disturbances. Vide "*Physiology of Nervous System*," page 156.

to record, carefully, all new evidence that may come to hand on this subject. The cases to which I now invite the reader's attention have come under my care during the last few years, and are part of those comprised in my Essay on "Injuries to the Head, and their Effects," to which the Astley Cooper Prize has recently been awarded. This Essay is now the sole property of Guy's Hospital, and is deposited in the library of that Institution. The trustees of the prize have, however, with much liberality, given me permission to publish such parts of it as I might think desirable, of which permission I now avail myself.

Contused Laceration of the left Eyebrow—Immediate symptoms of injury to the Frontal Nerve—Failure of the left Eye within seven weeks of the accident—Photopsiæ, but no pain in the globe—Subsequent failure of the other Eye—Deafness—White Atrophy of the Optic Nerves—Patches of absorbed Choroid in the left Eye.

Moses Isaacs, æt. 37, married; a steady, temperate man, with a large family. He came over from Melbourne in order to have advice as to his sight, being almost blind. On September 24, 1863, he was admitted at Moorfields, under my care. The following was his statement:—"I was standing one day in the High Street, Melbourne, when a man, in a passion with another man, in mistake struck me with an iron bar, a violent blow on the forehead. I was felled to the ground and was taken up insensible, and remained quite stupid for a couple of hours. The wound bled very much. A surgeon dressed it, and I soon got well. Seven weeks after this I was driving a cart near Forest Creek, when suddenly I found that I could not see, and was obliged to get another man to take care of the cart. My left eye was now quite blind; it had been gradually failing ever since the accident. After this my left eye never improved, and my right eye got worse and worse. I kept at work for some months longer; but, getting nearly blind, I was advised to come to England. When I sailed I could still see a little, but whilst crossing the equator in very hot weather, my sight quite left me. Still, I can see a little now and then with the right eye, and this very morning I walked a quarter of an hour in the

street alone, and recognised two people." On being asked as to the pain he had after the accident, he said that it was very painful just over the eyebrow in the wound, "pricking and drawing like," and he added, "I had the same feeling on the top of the head where I had not been struck." The part which he pointed out as that in which this pain was felt was on the left side, about the middle of the parietal bone, and therefore over the distribution of the frontal nerve.

Further questioning elicited that he had not suffered from headache nor from vomiting; that he had been liable to occasional swimming feelings in his head before the injury, but had enjoyed excellent eyesight. Since the accident he had lost flesh very much, but this he attributed to his having been desponding about his eyes. During the last eight or nine months his hearing has been failing, and he is now deaf in both ears, so that it is necessary to shout loudly. His left ear failed first, and is the worse of the two. He has been much troubled with noises in the head, ringing, &c. Flashes of light have been very frequent, and he saw them early after the injury. He never had any material pain in the globes. He is very sensitive, both to heat and cold, as they make his eyes water, and cause flashes of light; on this account he dare not, he says, go near a fire. The senses of smell and taste he considers perfect. Memory good, and no paralysis of limbs, either as regards motion or sensation. The sensation in the two sides of the face seems equally good, but on the left forehead he is decidedly hyperæsthetic as regards slight pricks, &c., and flinches sooner than on the other. He says it sends a shooting pain to the scar. He complains of having had a good deal of pricking and itching in the scar.

The scar is an irregular one; it commences almost over the inner canthus, just on the supra-orbital ridge, and extends outwards and upwards across the eyebrow, nearly to the outer angle. It is conspicuous and the wound has clearly been a deep one. I fancied that the bone beneath was a little uneven, but could not be certain. There is no thickening, and but little tenderness about the scar.

When he came to me at the Moorfields Hospital, he could but just with the right distinguish dimly the outlines of the largest objects; with the other, he could not tell light from darkness. The pupils were large and nearly round; but the iris above the

pupils was much narrower than below it. Both pupils were extremely sluggish, almost motionless. In the evening I examined him by candle-light. His amount of vision was much as in the morning. He could just dimly make out large objects, but in walking across the floor he ran against chairs, &c. Aspect, decidedly amaurotic.

The pupils dilated with atropine very slowly, and but little. In the right, the choroid everywhere of a homogeneous orange red; no single vessels visible. Optic disc, abruptly margined and conspicuous; grey white; arteria centralis and lesser arteries not discoverable; vena centralis still of considerable though much reduced size. In the left, the condition of the optic disc was exactly similar to that of the right, and the choroid was of the same velvety orange red appearance. The only difference in the fundus in the two was in the presence of certain very abrupt patches in the left choroid. A patch of considerable size, with most abrupt margins, was seen near the yellow spot; the choroid was removed wholly, and the white sclerotic was seen with a central island of black pigment. Near to this were several other aggregations of black pigment in the choroid, but attended by much less absorption of choroidal tissue.

It is, I think, clear that the cause of the amaurosis in this case is central. However produced, the atrophy of the optic nerves has, in all probability, originated in changes at their central origin. Had the injury to the frontal nerve any influence in producing this? In the first place let it be admitted that the evidence is strongly in favour of the belief that the left frontal nerve really was contused and injured. Then next let it be noted that the left eye began to fail immediately after the injury, and that it failed sooner and much faster than its fellow. Let us remember that he had a referred pain in the vertex of the left side of head immediately after the accident, and that, even now, eighteen months after the accident, he has a degree of morbid sensitiveness to irritation in the supplied tract of skin. Nothing could be more positive than the man's own conviction that his blindness is the direct result of his injury; and, looking at the order of sequence of symptoms, there can be no wonder that such is his impression.

I do not know how to interpret the patches of disorganised choroid in the left eye. It is possible that they may have been

caused by the early and more active morbid processes induced. From their appearance, it is certain that they are of long standing. There is no choroidal disease at present in advance.

The man expresses himself as being quite certain that the eye-ball itself was not struck, and is quite clear that he could see well for some days after the accident.

His deafness is probably due to similar disease to that by which his eyes have been affected, *i.e.*, central changes inducing atrophy of nerves.

Gradual Blindness of the Left Eye after a Contused Wound of the Left Temple.

Cornelius Leggatt, a farrier, æt. 53, who came to me on account of an injury to his right eye, gave me the following history of the loss of his left one:—Both eyes were perfect until about 28 years ago, when he received a blow on the left temple from a stone. The wound bled very freely at the time, but quickly afterwards healed. The eye itself was not struck, and it was not blind, nor materially affected as regards its sight until some time afterwards. Within a few weeks he found the eye irritable, "watery, and weak, especially in cold weather." Gradually he lost sight, and in about two years afterwards was quite blind. He attended the Moorfields Hospital, and was told that nothing could be done. He never had any pain in it, nor any photopsiæ.

At present he has no perception of light with the left eye. The pupil is round, rather larger than the other, and quite motionless. The iris is tremulous, as if the lens were displaced. The globe is a little turned outwards. There is not the least congestion of the globe, and the iris is brilliantly lustrous.

His other (the right) eye remained perfect until a blow received the other day.

Unfortunately I have mislaid my notes of ophthalmoscopic examination in this case, and also the man's address.

Large Scars in the Forehead from Lacerations five years ago—Loss of Sight for two years in the Left Eye—Atrophy of Optic Nerve.

Mrs. Simmons, from Aberystwith, æt. 70. Five years ago she was knocked down in the street, and had severe contusions on

the forehead. At present a large scar is visible in the middle of the forehead, extending towards the left side. There is another deep scar extending across the left eyebrow. She states that it was 11 weeks before the wounds healed. She did not notice any failure of sight until a year and a half ago, when her left eye began to fail. She can see well with the right eye. She complains of having "swimmings in her head." Pupils of equal size; both sluggish. She cannot count fingers with the left eye. Although atropine was three times introduced, the pupil scarcely dilated. The optic disc is grey, and there is a small irregular crescent on its inner side. It is slightly sunken. The veins are very large and dark. The choroidal pigment is accumulated in patches. The other eye appears normal, except, perhaps, that the nerve is a little cupped; but in this the effect of atropine has been but very slight. She can see well with this eye, but as she cannot read it is difficult to test her sight accurately. There is no increased tension of either globe, and the general signs of glaucoma are wanting. She has had no pain in the globes. The cupping is rather that of atrophy than of glaucomatous pressure.

Injury to the Right Frontal Region, and Contusion of the Face, followed by Atrophy of the Lachrymal Gland and Absorption of Fat from the Orbit—Much Impairment of Sight for a time, but subsequently recovered from.

The subject of the following case was under the care of my senior colleague, Mr. Dixon, who kindly gave me permission to investigate and record it.

Johnson Smith, a stout, florid man, æt. 42, came to the Moorfields Hospital on March 23, 1863. His face presented the singular condition represented in stereoscopic portrait No. 31 in my collection.

On New Year's Day, 1863, he fell from a ship's stage, 25 feet, and was taken up stunned. The accident occurred at seven in the morning. He was taken to the hospital, and until ten o'clock in the same morning was quite insensible. He then regained sensibility, and soon recovered. He left the hospital in nine days, quite well, except that the wound in the forehead was not entirely healed. In 18 days he returned to work. The right side of his face was, according to his statement, very

much bruised, but no bones were broken, and the only laceration was the one on his right forehead, of which the scar remains. It is an inch and a half long, over the outer half of the right eyebrow, and an inch and a half above it. Its inner end does not reach so far inwards as the middle of the eyebrow. It is now perfectly sound, and, according to his statement, the wound healed kindly, and very quickly. He appears to have had a severe contusion. He feels sure that his sight has got much worse during the last few weeks. He has a good deal of writing to do in the evenings, and his sight has failed so much that he cannot see his lines. He is sure that before the accident he could see with both, but was, indeed, always accustomed to use his right eye most, as his left was somewhat defective. After dilating the pupils with atropine, I found that there was in the left eye (the uninjured one) a considerable posterior synechia at the inner and lower part. At the same part, behind the iris, were also some considerable opaque bands, extending into the vitreous at the ciliary region. These had been quite concealed whilst the pupil was contracted. They must, I think, have resulted from some former disease, and probably had nothing to do with the recent accident.

With the ophthalmoscope nothing abnormal was noticed in the fundus of the right eye. As to swelling of the orbital tissues from the blow, he states that for several days his eye was swollen up, and the lids level with the cheek. It was subsequently very black. At the end of the nine days, however, when he left the hospital the swelling had disappeared, and, according to his statement (probably erroneous) the eye was already sunken somewhat.

Present state.—The right eye is very considerably sunken in the orbit. There is a deep hollow under the superior orbital ridge, into which the finger can be passed. No lachrymal gland can be felt. There is also a certain amount of hollowness in the lower part of the orbit, but not so much as to be very perceptible. The pupils are of equal size, and the left is fairly mobile; the right is less so. He can read No. 4 with the right eye. With the left he can with difficulty read No. 19.

August, 1863.—He can now read No. 1 with the right eye, and can see anything in distance perfectly. With the left he can only read No. 16, and cannot tell the time by the distant

clock. It is possible that he may have had some former injury to this eye. It is not improved by cataract lens.

He has very greatly improved in sight whilst taking pot. iod. gr. iij. ter die. The condition of the orbital tissues is just as it was. There is still the great hollow above the globe. He is in excellent health.

Blindness of one Eye, and Liability to Epilepsy one month after Contusion of the Head. Query.—A malingerer.

Thomas Osborne, a fruit hawker, æt. 18, had an altercation in the street with an Irish woman, who seized his pint measure from his stall and beat him violently about the head with it. He was stunned, and had severe scalp lacerations. He was carried into the hospital in a state of partial insensibility. There was a wound over the left parietal bone several inches in length. He lost a large quantity of blood. Whilst in the hospital nothing particular shewed itself. The wound healed well, and he was discharged in a fortnight. The injuries were received on September 13. On October 6, 1864, I was requested to examine him, as he could not see with his left eye. It now appeared that on two occasions since his discharge from the hospital he had had epileptic fits. One of these occurred in the police court, probably under much excitement, and the other a week later.

October 6.—The lad looks quite well; he has no squint, and nothing abnormal is to be seen in either eye. The pupils are of equal size, and act well. He avers that he can see very little with the left eye, only enough to distinguish very large objects. He sees the "blur of a candle flame," but cannot distinguish it clearly. Sees better from the outer corner of the eye. I tried him by placing a strong prism before his sound eye, both being open. He said he could see only one object, and persisted in his statement.

His account is, that he first found that his eye was failing on the second day after his leaving the hospital. Whilst in the hospital he could see as well with the one as the other. It has got much worse since he first found out it was failing. He complains much of headache, and says he is "dizzified and nervous." He says that the pain in his head will not let him sleep, and that he is so weak he cannot work. His smell and

hearing are good; he complains of a humming noise in both ears. There is a large recently formed scar, crossing outwards from near the middle of the scalp on the left side. There is a little thickening about the part, and he flinches much on the slightest examination of it.

His bearing and manner of stating his symptoms are such as to strongly suggest the suspicion of malingering. I tried him repeatedly with prisms and coloured glasses, but never could get him to admit that he saw double. He, however, several times, when I put a strongly coloured glass before the sound eye, asserted that the candle looked all right, and was not coloured; but this may have been from sheer stupidity. I do not, however, suppose that it was. The pupil dilated well with atropine, and on examination with the ophthalmoscope I could not detect the slightest deviation from the healthy state.

On covering the sound eye he said he could only see a glimmer of a candle placed in front of him; but when I used coloured glass, he would not admit that this glimmer was altered. On several occasions he was evidently considering what he should reply to my questions. Once I noticed him winking the lids of the left eye with great rapidity, and clearly by effort. Although, as stated, the prism test failed to detect deception, yet I am still much inclined to think that his present symptoms, epilepsy, headache, and blindness, are all feigned.

The above notes were written out a year ago. I have not seen the lad since.

(To be concluded.)

LOSS OF THE LEFT EYE FROM THE LODGEMENT WITHIN IT OF A PORTION OF A GUN CAP.—INFLAMMATION OF THE STUMP MORE THAN SEVEN YEARS AFTER THE INJURY, FOLLOWED BY SYMPATHETIC OPHTHALMIA OF THE RIGHT EYE.—CLINICAL REMARKS.

By George Lawson, F.R.C.S.,

Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and to the Middlesex Hospital.

George S., æt. 33, by trade a confectioner. Eight years ago, by way of amusement, he was exploding some percussion caps by striking them with a hammer on a stone. The caps were cheap ones, which he had procured at what he called a cheap Birmingham shop, in the country, at the price of 25 for a penny. Whilst so engaged a fragment of one of the percussion caps flew into the left eye, and there lodged itself.

The eye inflamed and suppurated, and, after much suffering, shrunk to a mere stump; but the foreign body, as far as he knew, still remained in the shrunken remnant of the eye. From that date, for more than seven years, he was completely free from pain; the lost eye gave him no inconvenience. The sight of the right eye was remarkably good, for he was a crack shot at long distances in the volunteer company to which he was attached.

Last February, for the first time, the stump of the left eye became painful and inflamed, and soon afterwards he noticed the sight of the right eye was foggy, but he suffered no pain in it. This attack continued about three weeks, the sight of the right eye becoming rapidly worse. Under treatment he recovered, but the sight of the uninjured eye was much impaired. Since then the stump has been repeatedly inflamed, and with each attack the right eye has sympathized, and on one occasion became acutely affected and very painful.

After each attack the sight of the right eye was materially injured, and although all irritation would for a time subside under treatment, yet he never regained any of the vision he had lost.

On September 22nd he applied to the Royal London Ophthalmic Hospital. The following was the state of the two eyes.

Left Eye.—A shrunken and contracted stump, inflamed, and looking red, and irritable. Through the centre of some cicatrix tissue, which occupied the place of the cornea, was projecting a metallic point, evidently the extremity of the piece of the percussion cap which had been so long lodged in the eye, and which was now making its way to the surface.

Right Eye.—Slightly pink and irritable. The cornea clear. The pupil fully the normal size, but fixed and completely adherent to the lens capsule, and the pupillary area of the capsule of the lens covered with a film of lymph. The striation of the iris very indistinct, and the anterior chamber very shallow, the plane of the iris having been approximated towards the cornea.

He is unable to read 20 of Jaeger's test types or to count fingers. He can only just make out the hand when held before his eye, or distinguish any large object in front of him.

He was at once admitted into the Hospital, and I removed the stump of the lost eye. On making a section of it, the piece of the percussion cap was seen to have made its way towards the surface, and was projecting by one of its edges through the cicatrix tissue in the front part of the stump.

The deep tissues of the eye were all so matted together as not to be recognised.

The man made a good recovery from the operation, and will continue to attend the Hospital as an out-patient. The right eye has decidedly improved. It is less irritable, and the pink appearance has disappeared. No amendment, however, can take place in the vision until some operation has been performed, as the pupil is bound down to the lens capsule, and the pupillary area is blocked up with lymph.

Clinical Remarks.—This case is a sad, but a very instructive one. It illustrates the danger of allowing a foreign body to remain in the eye, even though the eye for all useful purposes has been destroyed by it. Sooner or later it is certain to be a source of pain and trouble to the patient, and to induce an inflammation in the injured eye, which is peculiarly liable to excite a sympathetic ophthalmia in the sound one. In this patient there are some points worthy of notice.

1st. The injury was caused by the explosion of a cheap percussion cap. Unfortunately these cheap caps are made of an

inexpensive and brittle alloy, and scales of them often become detached during explosion, and fly off with such violence as to lodge themselves in any portion of the body with which they come in contact. In nearly all the eyes which are destroyed by percussion caps, and they are many, it will be found on inquiry, that the caps used were the toy, or the cheap ones, such as children buy for their play guns, or such as are employed in firing at targets in the streets for nuts.

2nd. The length of time which a foreign body has remained embedded in an eye, is no guarantee that the danger of its presence has passed. In this patient over seven years elapsed before it began to give trouble, but when the stump of the lost eye became inflamed, the sound eye was speedily involved in that terrible disease, now so well known as sympathetic ophthalmia.

3rd. The sound eye in this patient was sympathetically affected without its suffering in the first attack any pain as a warning of its presence, and this is one of the peculiarities of the disease. It will often creep on unheeded by the patient, the impairment of the sight being so gradual as at first not to draw sufficient attention to it; and it frequently happens that the disease is fully established before it comes under the surgeon's care.

Since the source of irritation, the stump of the lost eye, with the foreign body within it, has been removed, the other eye has decidedly improved.

Time must now be allowed for all irritation to completely subside, and after an interval of some months if the eye continues so long in a quiescent state, an operation will be performed to establish a new pupil, and if the lens on the removal of a portion of the iris, should be found cataractous, it will be removed. There is yet a fair prospect that the man may recover a useful eye.

TREATMENT OF TWO CASES OF PTERYGIUM BY TRANSPLANTATION.

By George Lawson, F.R.C.S.,

Assistant-Surgeon to the Royal London Ophthalmic and the Middlesex Hospitals.

In the two following cases I treated the pteryguim in each eye by transplantation, after the manner recommended by Desmarres.

The operation is easily performed, and its results, as far as I am able to judge from the time the patients were under my care, very satisfactory.

The lids being separated by a spring speculum, I seized with a pair of finely toothed forceps the extremity of the pteryguim close to the cornea, and then with a pair of fine scissors clipped away carefully and accurately its attachment to the cornea, still holding the pteryguim between the blades of the forceps. I then made a cut with the scissors through the conjunctiva, along its upper border, and then another through the same membrane along its lower border, in the direction of the dotted lines in the diagram No. 1. At the point of the lower free cut edge of the conjunctiva, to which I wished to insert the apex of the pteryguim, I made a small snip with the points

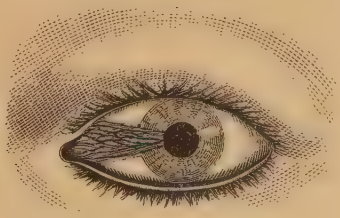


Fig. 1.

of the scissors, and then fastened it *in situ* with a single suture of very fine thread, never having released my hold

of the pteryguim with the forceps until the operation was completed (fig. 2). I had an opportunity of seeing each case about one month after the operation, and the pteryguim in each eye in its new situation was gradually shrink-

Fig. 2.

ing. In cutting away the insertion of the pteryguim from the cornea, a Beer's knife or a fine scalpel might be used to dissect it off, in preference to clipping it away from its attachment with a pair of scissors.

The advantages of this mode of treatment are:—

1st. There is no loss of conjunctival tissue.

2nd. The pteryguim does not recur.

In its new site it is reported to gradually shrink, and to cease to give the patient any annoyance. I have on several occasions abscised a pteryguim, and brought together the edges of the conjunctiva as accurately as possible, but certainly in two instances I have had to repeat the operation more than once, and in one case the cicatrice which followed the loss of the conjunctiva on the inner side of the eye caused a considerable limitation of its movement outwards.

Case I.—William G. came under my care on August 29th of this year. For the last eight or nine years he had been engaged in mining in Australia. About four years ago he had an attack of purulent ophthalmia, and he dates the commencement of his present affection of the eyes from that period.

He has now a pteryguim in each eye.

In the *left eye* it is very thick and fleshy, and extends quite up to the level of the margin of the pupil, when not dilated.

In the *right eye* it is less developed, but still very conspicuous. It has encroached upon the cornea, but has not reached as far as the pupillary edge. In each eye I transplanted the pteryguim downwards in the manner just described. Slight conjunctival redness followed, but in four days the suture was removed, and the patient left the hospital.

Case II.—George W., æt. 53, came to the hospital on August 22nd of this year, suffering from a pteryguim in each eye. His employment is on the water. He is constantly engaged at work on the oyster beds at Colchester.

In the *right eye* the pteryguim is large, and extends over the cornea quite up to opposite the edge of the pupil.

In the *left eye* it is much smaller, and only encroaches slightly on the cornea.

In this man I also transplanted the pteryguim in each eye downwards. Very little irritation followed, and he left the hospital on the 28th.

About one month afterwards I saw each of these men. The pteryguim in each eye was much shrunken. They were pleased with the result, and had experienced no inconvenience from the operation.

A CASE OF PYRAMIDAL CATARACT, WITH MICROSCOPIC EXAMINATION (BY PROFESSOR C. SCHWEIGGER) OF THE LENS AFTER EXTRACTION.

By A. Samelson, M.D. (Manchester.)

John Procter, hooker, 23 years old, pock-marked, presents himself with a cataract in his left eye. The iris of this eye appears greenish-grey, whilst that of the sound right eye is of a light blue colour. The pupils of both eyes act equally well. The left eye, in addition to the cataract, is affected with a faint nebulous opacity occupying the centre of the cornea, a moderate lachrymal obstruction, and a slight divergent squint. In respect to the latter, we meet with this peculiarity, that the internal rectus, far from being weakened by its permanent extension, is able to carry the cornea so deeply into the great angle as to hide almost the entire inner third behind the caruncula, whereas in the right eye, under the greatest effort of its inner rectus, the corneal margin remains nearly a line distant from the caruncula.

The cataract, said to have been left behind by "the small-pox and measles" when the patient was three years of age, presents the following characteristics. A white cone, very conspicuous, much like a minute mamilla, of an apparent depth of $1\frac{1}{2}'''$ and of smooth surface, is seen to project through the pupil into the anterior chamber; in profile, the apex seems almost to touch the posterior surface of the cornea. There appears to be an interval between the base of the little pyramid and the sort of wider socle, on which it seems to rest with a short and narrow pivot. The surface of this socle is not of the uniform smoothness and whiteness of the pyramid, but has an irregularly burrowed, or, as it were, curdled appearance.

The patient distinguishes the back from the palm of a hand, and, when turned from the light, counts fingers and tells their movements; he sees better when the object is placed toward his left side. After the pupil has been well dilated by atropine he is just able to see the ear or nose of a person standing before him, but there also becomes visible a black zone, about $1\frac{1}{2}'''$ in breadth, surrounding the "socle," the circumference of which is seen o be irregularly serrated, and a minute knob here

and there projecting from it; the short pedicle intervening between it and the pyramid is now more plainly discerned. On looking into the pupil from the right side, at an angle of about 15°, we perceive a dull red glare coming from the fundus, of the details of which, however, nothing can be made out. The case was presented to the Manchester Medical Society.

On the 19th October, 1864, the cataract was removed by linear extraction, the incision being made at the outer side of the cornea. I performed also a small iridectomy, on the one hand to facilitate the exit, if possible, of the whole of the cataract, on the other, to obviate inflammatory consequences. These I was led to apprehend by the discoloration of the iris, as pointing, in my view, to bygone irido-choroiditis, which, though it left the pupil free, might have occurred contemporaneous with the formation of the cataract, and damaged the deeper parts of the eye sufficiently to account for the existing degradation of sight. For, this, after the action of the mydriatic, appeared to me disproportionate, unless it was to be brought home entirely to the long exclusion of the eye.

I succeeded in bringing away the cataractous pyramid entire, together with a number of small, irregular particles, obviously fragments of the "socle." The pupil, however, even in its centre, did not appear normally black, and there remained, skirting the pupillary margin, a greyish-white ring of cataractous substance, varying, but at its widest not exceeding 1‴, in width. This, in course of time, has almost entirely become absorbed; but the patient's vision is not in the least improved by the operation; he is, however, quit of a very bad disfigurement. The lachrymal obstruction had been removed without difficulty by Weber's method. The divergence was not attempted.

Ophthalmoscopic examination, somewhat hampered by the corneal haze, discloses, besides slight pigmentary anomalies in the choroid, an optic disc presenting all the characteristics of advanced atrophy: the lamina cribrosa very distinct, the remaining arterial twigs most attenuated, that blotchy appearance of the likewise scanty veins, so common in cases of inveterate squint with its attendant amblyopia ex anopsia; and the presence of the retina is indicated by a small number of its vessels only.

The morbid specimen was seen with interest by Professor

von Graefe, and at his request, by Prof. Schweigger subjected to microscopical examination, of the results of which the latter was kind enough to send me the following account:—

"The cone, measuring about 2 mm. in height, and at its base about $1\frac{1}{2}$ mm. in width, under a low magnifying power presents a large number of fine longitudinal folds, and at its base merges into an amply-folded glass membrane. This, though greatly thinned, is yet, by the presence of many disaggregated remnants of the intra-capsular cells, characterised as the atrophied anterior capsule. In conjunction with the latter we find a thick mass, which, under a higher magnifying power, appears striped and stippled, becomes much clearer when acted on by diluted acids, and, together with a good deal of cholestearine and other spear-shaped crystals so frequently occurring in metamorphosed lenses of the kind, exhibits distinct indications of a delicate glass membrane of recent formation. Longitudinal sections of the (previously hardened) cone* show it up to the apex to consist of a mass striated parallelly to the surface of the lens, and quite of the structure habitually met with in transverse sections of capsular cataracts. The striæ which can easily be torn out into thin lamellæ, on the smooth surface of the cone cohere intimately with each other, and thus present the semblance of a delicate membrane which, however, cannot be isolated as such. Nearly corresponding to the base of the cone we discover a fatty or calcareous mass, further on a striated texture, and further again more amorphous masses of the same nature as the loose spherical fragments which the bottle contained in addition to the cone."

"To the question, then, which here chiefly concerns us, whether the cone was interior or exterior to the capsule, we do not obtain a satisfactory answer; in the transverse sections, to which we had to look for a solution, no trace whatever of a capsule was found. Nevertheless, it appears to me highly probable that the whole mass was invested with a thin, atrophied anterior capsule, inseparably coherent with the surface of the cone."

* Professor Schweigger retains beautiful specimens of these sections which he showed us at Berlin, on his return from his journey to London, where he had undertaken the examination.

OBSERVATIONS ON DEFECTS OF SIGHT IN DISEASES OF THE NERVOUS SYSTEM.

By J. Hughlings Jackson, M.D.,

Assistant Physician to the National Hospital for Epilepsy and Paralysis, and to the London Hospital.

(Continued from Page 446.)

It is very difficult to comment on cases of amaurosis in a Journal devoted to the special consideration of defects of sight without running the risk of being too diffuse. It is not easy to find a satisfactory arrangement. Some of the cases I relate have very wide bearings in physiology, pathology, and clinical medicine. An arbitrary arrangement which exaggerates one aspect of the cases would be really to cover up disorder. The last recited case refuses to come under any one particular heading. Although for convenience of space we speak of it as a case of amaurosis, we must also think of it as:—(1) A case of syphilitic disease of connective tissue. In this plane of thought the fact that the connective tissue disease is in the pia mater of the hemisphere is comparatively unimportant. I suppose it would not differ in any essential particular from a cellular node of the buttock, or muscular node of the tongue, or what I may call a "liver node." (2.) As a case of disease of the hemisphere. Here again, so far as the symptoms due to loss of a certain part of the hemisphere go, it is of no importance whether its substance was diminished by syphilis, cancer, tubercle, or softening. The most important, or at least the most striking fact, was that with disease of this part of the right hemisphere speech was not affected. (3.) As a case in which a "foreign body," viz., a syphilitic tumour, had produced general disturbance in the brain leading to amaurosis, &c. Here a consideration of the nature of the disease comes to be of importance, as, so far as I know,

mere softening does not excite such changes; but now its position is of much less consequence,—as regards the amaurosis at least. (4.) The clinical history suggests points of very great importance. It would have been thought, before Helmholtz discovered the ophthalmoscope, that, there being syphilitic disease of the connective tissue of the hemisphere, the amaurosis would be due to syphilitic disease of the choroid. The case corrects this easy way of concluding.*

I think it but right to make the above statement, but the reader will no doubt gladly excuse me for not dwelling on all the points in it. To speak of the cases as widely as one ought to think of them, would be to write the greater part of a treatise on diseases of the nervous system. In the former part of this paper I related cases, and made remarks, simply with the object of shewing the importance of the ophthalmoscope to the physician in his investigations of cases of disease of the nervous system. In this part I shall chiefly consider amaurosis as it occurs from disease of the hemisphere.

* This lateral way of thinking is not safe. I have had, during the last six months, two patients under my care for paralysis of several cranial nerves. The cause during life was clearly syphilis; and there was found, after death, syphilitic disease of the (arteries of) liver, and around the arteries of the brain. Now each of these patients had aphonia. The conclusion that they had actual disease of the larynx itself was the readiest and easiest way of explaining it. The case shows that the use of the laryngoscope will help the physician as the ophthalmoscope does. Dr. Morell Mackenzie found, on laryngoscopic examination, that the larynx was healthy, and that the aphonia was due to disturbance of its function, viz., to paralysis of one vocal cord. He was bold enough also to say that the paralysis was of the crico-arytenoidens porticus muscle. The larynx in each case was carefully examined by Mr. Frederick Mackenzie, and nothing was found except partial atrophy of the above-named muscle. A rational explanation of this functional disorder was given by the autopsy. Again, in defects of sight let us say in hemiplegia with Bright's disease, without the opthalmoscope we might infer that the changes were those characteristic of Bright's disease. So they were in Case XI. But if we examine we may find, as in Case XII, that they depend on neuritis from disease of the hemisphere, the Bright's disease being connected with the failure of sight in but a roundabout away.

I will, however, first say a few words on paralysis of the third nerve, which in the last case I have related complicated the amaurosis and hemiplegia from disease of the hemisphere. Paralysis of this nerve is a very rare complication of hemiplegia, and this is the only instance I can call to mind in which I have found it with hemiplegia and double amaurosis. I have only once seen hemiplegia on the right side, with loss of speech, complicated with paralysis of the third nerve. Disease of the motor tract, above the crus cerebri, and, *à fortiori*, disease of the hemisphere, do not produce complete paralysis of any cranial nerve. When there is paralysis of a cranial nerve and hemiplegia, with double amaurosis or loss of speech, the probability is that there are two lesions. We know that in disease of the inner and under part of the crus cerebri* we have hemiplegia on one side, and paralysis of the third nerve on the other. I thought during the life of the patient that the disease was so placed in him. I imagined that there was disease of the right crus, and that this disease produced amaurosis, by involving first the optic tract of its own side, and then by extension the one of the other side. Further experience makes me more cautious in concluding that there is disease of the crus when these two symptoms are found together. If they come on together suddenly, I should feel pretty confident that the crus was diseased, but in chronic cases, where one follows the other, the diagnosis of two lesions, one of the hemisphere and one of the trunk of the third nerve, would be just as likely. Moreover, I have doubts whether disease of the optic tracts would produce the ophthalmoscopic signs of neuritis. I had under my observation for some time a case of hemiplegia, with hemiopia (Wollaston's disease). There was besides paralysis of each third nerve, although on one side not complete. This patient died, but I could not procure an autopsy. During the stage of hemiopia, when the dis-

* A most valuable paper on disease of this part of the brain has been published by Dr. Hermann Weber, Med. Chir. Trans. 1863.

ease of the optic nervous system must have been of one optic tract only, the discs looked quite normal; but before the patient died he became blind, and then the discs were quite white. There was however no trace of neuritis. The disease here must have been of or near to the crura cerebri.

In Zehender's Ophthalmic Journal, August, 1864, is related a case* which is of great interest in this connection.

The great interest Case XIII. had for me, however, when I had made the autopsy, was as one of disease, involving, among others, a particular convolution, the fellow of which in the left hemisphere is, according to M. Broca, the seat of the faculty of articulate language. Although I have greater (and increasing) doubts than I had as to the propriety of using the term "faculty" at all, and greater doubts still as to the possibility of locating any faculty, if there be one, in a particular convolution, I believe that M. Broca has conferred a great boon by introducing method in an inquiry which had been rather confused.

At the time when I made the autopsy I was much struck by finding amaurosis instead, as it were, of loss of speech. I have several times since found disease in this region (on the right side), with amaurosis, and with no defect of speech of any sort. I use the term "region," as the disease has never been very local, and, moreover, has not

* It was there reported in reference to a question I had asked as to the comparative frequency of hemiplegia on the right or left with amaurosis. In asking the questions I ought to have explained that I meant by "amaurosis," blindness of both eyes, due to optic neuritis. I may however say that, except in the case of hemiopia just mentioned in which, no doubt, there was disease of one of the crura cerebri and not of the hemisphere, I have not seen any other form of atrophy of the optic nerves with hemiplegia. Dr. Laqueur's interesting case therefore does not bear on the question I have asked. In other points of view his report and remarks are of the very highest value. The case looks like one of syphilitic disease of the arteries of the brain. This would account for the embolism, which, as he suggests, is the most likely explanation of the hemiplegia on the right side, with the defect of speech.

always been limited to the surface, so as to be easily defined as regards the position of convolutions. In several cases related in this paper, it will be found that the (right) fellow of M. Broca's (left) convolution was only one of several involved.

The simultaneous study of amaurosis and defects of speech has been thus forced on me. Apart from any theory* whatever, there is a great advantage in this, and I strongly urge it on the consideration of ophthalmic surgeons. It is simply impossible for a physician to do otherwise in working at diseases of the hemisphere. I may therefore be excused if I dwell more on defects of speech than seems *primâ facie* reasonable in a Journal of Ophthamology. For my part, I think Case XII. shews that ophthalmic surgeons cannot avoid studying the two symptoms, for there the lesion which caused at once defect of speech, next, insidiously, gave rise to the secondary symptom, neuritis.

Although I have grave doubts as to the localisation, or even as to the existence of any such faculty as that of language or articulate language, it is not contradictory to say that, so far as my experience goes, disease in only one region of the brain causes defect of speech. Facts force me to this conclusion. When I was first studying this point, it occurred to me that there might be particular parts of the hemisphere, disease of which produced amaurosis. A little thought, however, showed me that there was abundant evidence to prove that amaurosis resulted, not only from disease of widely different parts of the hemisphere, but even from other divisions of the nervous system, as the cerebellum, and also from tumours at the base of the skull. Yet in the cases of disease of the hemisphere that I have related, the damage has been rather wide-spread. Whilst, then, I still hold that the present evidence points to the conclusion that disease of any part of the hemisphere is

* I say apart from any theory, but I shall give my facts as I gathered them, viz., when my mind was full of the question as to the localisation of faculties.

associated with neuritis, I would raise the question whether, from more cases, and more precise localisation of the disease, we might not find this gross conclusion to be only partly the truth. Although it is, I think, impossible to fix on any one region of the hemisphere, as the seat of damage in neuritis, I think it not impossible that damage of some convolutions may be more likely to give rise to it than of others. If it were really so, the pathology, or rather the physiology of amaurosis, would still differ widely from that of loss or defect of speech, in which but one region of the brain appears to be affected.

I have been struck with the greater frequency with which I have found optic neuritis, with hemiplegia on the left than on the right side.

The facts I have observed go to prove that disease in a certain region of the left hemisphere produces loss of speech, and other facts go to shew that disease in the corresponding part on the right does not affect speech at all. Having found disease here on the right, with amaurosis, several times, I have been led to pay much attention to this part, and I was at one time sliding into the belief that disease of it might be more likely to induce amaurosis than disease of the other parts of the right hemisphere. It will, however, at once occur to every one that it would not be fair to conclude that disease of this part was more likely to produce amaurosis, because in my cases of amaurosis it might be the part most often involved. In the first place, I am more likely, as a physician, to be consulted in those cases of amaurosis from disease of the hemisphere in which this region is involved, and simply for this reason, that disease here is liable to involve the corpus striatum, and thus to produce hemiplegia, a symptom for which a physician is consulted. Again, I am under the impression that disease more often occurs in this position than it does in other parts of the hemisphere. Certainly effusions of blood do, as the region of the corpus striatum is very vascular.

In all the cases of neuritis in which I have made a

post-mortem examination, there always has been found notable organic disease in the head. But as I have repeatedly said, from this symptom, with or without headache and vomiting, we cannot during life predict where the intracranial disease is, nor what it is. If, however, there is hemiplegia besides, or, as I think, unilateral epileptiform convulsions, we have strong evidence that the amaurosis depends on disease of the hemisphere. The hemiplegia may precede or follow the amaurosis; it may be passing or permanent, but in all the cases in which it had been in any way present during life, I have found disease* of the hemisphere *post-mortem*.

Of course, if the hemiplegia be on the right side, the disease of the hemisphere is on the left, and if on the right the left hemisphere is diseased. Now it is only when the disease of the corpus striatum involves the hemisphere near it that loss of speech attends hemiplegia, and so it is, I think, only under such circumstances that amaurosis comes on after hemiplegia has been some time established, or follows it when it has passed off.† The association of hemi-

* I have seen, however, a case of amaurosis in the practice of Mr. Hutchinson in which the existence of slight weakness on the left side lead me incorrectly to diagnose disease of the opposite hemisphere. At the autopsy we found no disease of the hemispheres. There was a tumour at the base which grew so as to press on the left crus cerebelli, thus producing, as we know from Dr. Brown-Séquard's researches, disease here sometimes does, paralysis on the same side as the lesion. In such cases (as there was in Mr. Hutchinson's patient, some time after my erroneous diagnosis), paralysis of one or more of the cranial nerves generally comes on to show that the disease is at the base. The fifth nerve was involved. [It unsettles one's mind very much to see many cases. Since writing the last few sentences I have called to mind a case I saw in Guy's Hospital, in which, with neuritis, there had been paralysis of one fifth nerve, and yet at the autopsy the disease—a large tumour—was betwixt the hemisphere and the fifth nerve, and the pons crura, &c., seemed to be quite healthy.]

† When I afterwards speak of epileptic hemiplegia I shall point out that disease on the surface of the brain is attended with hemiplegia, although the obvious disease does not involve the motor tract. But in the text I am speaking of cases in which it may actually damage the motor tract, and the convolutions near it. So far as I know, extensive disease of the hemisphere above the ventricles need not produce any local paralysis, but only general weakness.

plegia with loss of speech or with amaurosis is then, as it were, an accidental one. It is true that it is an accident that nearly always happens with loss of speech, and not very unfrequently with amaurosis. Still the hemisphere may be alone affected, and then we have loss of speech only, or amaurosis only, according to the position, and, in the case of amaurosis, according to the position of the disease and other circumstances to be presently alluded to. As I have said also, loss of speech comes on at the same time as the hemiplegia—follows at once upon the damage—but amaurosis never comes on suddenly, as the result of injury to any part of the hemisphere. It will be seen then that the hemiplegia is to the ophthalmic surgeon a useful symptom in pointing out the position of disease in a given case, but it does not point to any peculiarity in the amaurosis itself, as just the same kind of amaurosis follows disease in very different positions. A utilitarian might then say that the hemiplegia is a symptom of little use in scientific investigation as the particular position of damage to the hemisphere seems almost to be a thing of no consequence, so far as its power of producing amaurosis is concerned.

Most people, however, will agree that it is always useful to recognise a fact whether its immediate importance seems to be great or little. But I wish just now to urge the study of hemiplegia with amaurosis, chiefly in order to furnish negative evidence as regards speech. During life the evidence can only have an approximative value, but in order that our autopsies may be of use, the other symptoms associated with amaurosis during life must be studied carefully. I think much good will be done if physicians and ophthalmologists make this a common ground of work. Case XIV. shows that if the physician does not examine the eyes, he will frequently lose a most important symptom. There does not appear to be great risk that an ophthalmologist, in attending to amaurosis, would overlook any considerable defects of speech, as these phenomena obtrude themselves. It is quite otherwise as regards defects of

sight, with total, or almost total, loss of speech, as Case XII. shews. There was nothing to direct my attention to the patient's eyes. I examined them as a mere matter of routine.

Case XIV. should be compared and contrasted with Case XII. Each of the patients was brought to the hospital with hemiplegia on the right side and loss of speech, and each of them had old disease of the optic nerves which clearly did not depend on the recent disease of the hemisphere, but which, to my thinking, does invariably depend on intracranial disease of some sort. In each of these patients I felt sure there were two parts diseased.

We all know that if a patient has Bright's disease and hypertrophy of the left ventricle, and degenerated arteries, he is liable to effusions of blood in various parts of his brain. As a rule, they affect the regions of the corpus striatum or the thalamus opticus, and thus generally produce hemiplegia. If the effusion of blood be in the hemisphere away from the motor tract, the patient may only have what he calls a "fit," and soon be well again. It was quite clear that the left corpus striatum and the adjoining hemisphere had been recently affected in each of these cases, and in Case XII. the former attack of hemiplegia pointed to old damage of the same region. Now I confess that in Case XII. I almost expected to find disease of the right hemisphere, as well as of the left, although there had been no left hemiplegia to point to it. At the autopsy the right hemisphere was quite healthy, so that I received a check which prevented my sliding into the conclusion that disease of only the right hemisphere produced amaurosis, as disease of the left only seems to produce loss or defect of speech. The autopsy in Case XII. shews, so far as one case can shew, that disease of the left hemisphere may first disorder speech and then give rise to amaurosis. In Case XIV., there was also a history of an old attack, viz., of fits, and these were very likely due to disease of the

hemisphere at a previous period. Whether that disease was of the right or left hemisphere, there was no evidence to shew.

In stating the mere fact that there is loss of speech or amaurosis, with hemiplegia, it is very important to note whether the loss of speech came on at the same time as the hemiplegia, or before it or after it, and especially when there is amaurosis it is very important to make out whether it preceded or followed hemiplegia. It must be kept in mind that the coincidence of the two things, loss of speech and hemiplegia on the *left* side, does not imply that they both depend on disease of the right hemisphere, but more likely that there is disease on each side of the brain. A few months ago I made a *post-mortem* examination on a patient who had hemiplegia on the left side and loss of speech. The patient had been under the care of Dr. Begley in Hanwell, and to this gentleman I am indebted for particulars of the life history of the case. Although I should attach very little importance to the co-existence of these two symptoms when they came on at different times, as they did in this instance, I know that many would insist on such a case being recorded as an exception to the rule, that when speech is lost or is defective the hemiplegia is on the right side. If so, the exception proves the rule, as there was disease to the outer side of the left corpus striatum, and at the junction of the right corpus striatum with the right thalamus opticus. Similarly we cannot conclude, because a patient comes to us with hemiplegia on the right side, with defect of speech and amaurosis, that there is no disease of the right hemisphere as well as of the left. Case XV. shews the importance of investigating the relation of amaurosis to past symptoms most carefully. His case was superficially like that of Case XIV. except that he could talk well enough to give me, with his wife's help, a history to shew that the hemiplegia on the right and the amaurosis were not related. There can, I think, be little doubt but that at the first attack the clot

damaged the right corpus striatum, and thus gave rise to the hemiplegia on the left, and at the same time damaged the adjoining hemisphere, and thus rendered him liable to the amaurosis which followed. The second attack of hemiplegia with loss of speech was certainly due to damage of the left corpus striatum and its neighbouring hemisphere. Yet whilst I thus drew attention to the fact that in this instance the amaurosis did follow disease of the right hemisphere, Case XII., shews that it might have followed the disease on the left side. It may thus be said that the distinction is of no practical importance. It is always important to know as much of our cases as we can, and I have faith that the knowledge we obtain will serve us sometime, although its use may not be at first apparent.

I will now speak of other aspects of cases of hemiplegia with amaurosis, although in doing so I must, I fear, ecapitulate somewhat.

I have not yet made a *post-mortem* examination of a patient who had had neuritis without finding some obvious organic disease within the head, and I think the evidence goes to prove that in most cases this organic disease has existed for some time before it excited those changes, which gave rise to disease of the optic nerves. Now, in Cases like XII. and XV., the hemiplegia is valuable evidence as to the position of such organic disease, and in patients of that age, considering also the suddenness of the paralysis, it is some evidence as to its nature. They each had clearly bad attacks of apoplexy. Now sometimes, when but little of the motor tract is damaged, the hemiplegia may pass off nearly altogether, although of course there remains permanent damage to the brain. The patient, Case XII., got well enough to go to sea again, and had a second attack when he was upon the rigging, yet at the autopsy we found an apoplectic cyst* in his brain, the size of a walnut.

* Page 436 line 2, the word "free" should be "front."

A patient goes to an ophthalmic surgeon for amaurosis; he will very likely say that his defect of sight began in the midst of perfect health, although he may at that very time have the remains of a large clot of blood in his brain, as Case XII. had when he consulted Mr. Hutchinson. Although, as I have said, I should think that every patient who came to me for neuritis only would have some organic disease in his head, I could form no idea either as to the position of that disease nor as to its nature. The position of the disease might be guessed, but I know of no means of making a diagnosis.*

If I found that a patient with neuritis had had hemiplegia some time before, although he might have quite recovered from the paralysis I should think it extremely probable that the neuritis was a secondary symptom of the disease which had produced the hemiplegia. If the patient were a man of middle-age, and above all, if he had albuminuria, I should think that that disease was the result of apoplexy. When a patient ceases to be incapacitated by disease of the brain, he naturally thinks that he is well, and, indeed, he may be well enough for routine work, either of mind or body, when a great deal of the hemisphere is damaged. So far as we can tell, he may, indeed, keep well, but, as in Case XII., he is liable to have headache, neuritis, &c. And when he does suffer, it is clearly not because a certain quantity of nerve-fibres and nerve cells are destroyed, nor is there yet evidence to show that it is because the function of any particular part of the brain is lost. It seems to me that the patient suffers because he has "a foreign body" in his brain. And even this is scarcely a correct expression, as a patient may have a

* In recording Case II., p. 398, I could give no opinion as to the seat of the disease, and I could not form any until the weakness of the left arm came on, and even then I could only make the wide diagnosis of disease of the right hemisphere. Moreover, as the hemiplegia came on gradually and was never quite complete this diagnosis, remembering Mr. Hutchinson's case of tumour of the brain, see footnote to p. 57, could not have been quite certain.

large tumour in his brain, and yet not suffer at all. Strictly speaking, therefore, he does not suffer from the disease in his hemisphere, but is liable to suffer from it. In Case II. there is little doubt that the patient's deceptive recovery was not due to any retrocession of the primary disease, but to his having weathered the storm it had excited in his nervous system with—if I may use such an incoherent simile—only the loss of his sight.

Why one person should have extensive disease of hemisphere without amaurosis, nay, indeed, without any symptoms* of any sort, and why another should suffer; or again, why a person's brain should tolerate disease for a time, and then suffer, I do not know. Of the fact there can be no doubt whatever. Just as a person may have disease in his liver without any liver symptoms, so a person may have disease in his brain without any brain symptoms. Probably a healthy man has more brain than he uses, just as he has more liver or lung than he uses. I had a year ago under my care a patient who had one pleural cavity full of fluid. I told him he was seriously ill, but he did not think so; he "felt well," and went to

* The following is taken from one of our medical journals :—

"*Vast Abscess of the Brain, without Symptoms.*—M. Richet related in the Society of Surgery the case of a youth, aged 18, who applied as an out-patient at the St. Louis, on account of a purulent discharge from the ear. So little inconvenience did he feel from his ailment, that he was with difficulty persuaded to enter the hospital for treatment. With every appearance of excellent general health, he died suddenly next day, immediately after the occurrence of a convulsive paroxysm. At the autopsy the petrous bone was found diseased, but the dura mater covering it had not undergone any change. The cavity of the tympanum was filled with pus, which obtained its discharge both by the meatus and Eustachian tube. All the convolutions of the left hemisphere had become effaced, and a collection of pus occupied the whole of the sphenoidal and occipital lobes, the parietal lobe remaining alone intact. Very small abscesses were scattered throughout the parietal lobe. This patient had never manifested the slightest intellectual disturbance, and no symptom indicated the existence of cerebral lesion, when the pus, bursting into the lateral ventricle, caused instant death."—*Gazette Hebdomadaire*, No. 46.

his work as a drayman. He has been at work ever since, although the fluid has not disappeared even yet. Of course in the cases of amaurosis I relate I can only presume from general evidence that the cerebral disease has been latent. I might cite authorities to prove that in patients who die of other diseases than those of the nervous system, disease of the hemisphere is found *post mortem*. Dr. Rooke, of Greenwich, showed me the brain of a patient who had died of phthisis. There was a large abscess in one hemisphere. About a year ago I saw a *post mortem* at the London Hospital, on a patient who had abscess of the liver, and who had had no cerebral symptoms until the day before he died, when he had a slight convulsive seizure. Yet this patient had two or three large abscesses of some standing in one hemisphere. Of course when I say that disease of the hemisphere need not produce any symptoms, I exclude the motor tract, and—on the left side at least—the convolutions near it. A very small amount of disease of the motor tract would produce paralysis, just as a small amount of disease of the liver, involving the biliary duct, would produce jaundice. If the disease be away from the motor tract or biliary duct, there need be neither, as the case may be, paralysis nor jaundice.

Some at least of the cases show well that of the three symptoms of which I have spoken so much,—headache, vomiting, and amaurosis,—two may have existed for some time before the third came on, so as to actually incapacitate the patient. I have, at page 417, Case VI., alluded to the unfortunate end of cerebral cases, in which the early symptoms of pain in the head and vomiting led to their being called "bilious headaches," and which have been considered unimportant, except for the temporary distress they occasioned. Case XVI., Mr. Hogg's patient, also illustrates this. Of course there is no possible means of being absolutely sure that that patient's right hemisphere had been long diseased; but I think that it is almost certain that it had been diseased since early life. I cannot help thinking that

the disease we found had been an abscess resulting from disease in the ear.—See *Toynbee on Diseases of the Ear.*

In Case XVII, sight did not begin to fail until three months after the disease had begun to excite changes in the patient's brain. She had first severe pain in her head, for which she went to a general hospital; then defect of sight, for which she went to an ophthalmic hospital; and finally, epileptic seizures, for which she consulted me at the Hospital for Epilepsy and Paralysis. And it is very likely that the poor woman will go to some other institution—probably the workhouse—to die. Her case (and the sequel of Case II.) therefore, illustrate what I have written, page 439, on the difficulty of keeping patients under observation when their symptoms change. I have little doubt that my patient has had from first to last but one focus of disease, which has produced all her different symptoms. Mr. Carter, in a note to his translation of Zander on the Ophthalmoscope, says:—"In the out-patient room of the Hospital for the Paralysed and Epileptic, it would at any time be easy to find cases of partial or complete cerebral amaurosis, in which the cerebral symptoms do not by any means force themselves upon the observer." Mr. Carter's observation is quite correct, but an investigation of the history of such cases nearly always reveals other, and often very definite, brain symptoms. I think that the unilateral convulsions—in this case on the left side—point quite as certainly to disease of the opposite hemisphere as hemiplegia would have done. Besides there was a little paralysis following her attacks, in other words, she had epileptic hemiplegia. Case XVI was very much the same in its general features, but here the unilateral convulsions were more violent and more continuous, and therefore the epileptic hemiplegia was more decided. I have as little doubt that there is, in Case XVII, disease of the right hemisphere in the region of the right middle cerebral artery, as I had that there was in Case XVI. I have now under my care also a patient, Case XVIII, in whom it is certain that the hemi-

sphere is diseased. He has both partial paralysis and epileptiform seizures. In his case the paralysis is not due to the violence of the convulsions but the spasm begins in muscles that are already partially paralysed.

CASE XIV.—*Hemiplegia on the right side with loss of speech—Amaurosis—History of former Cerebral symptoms.*

Hannah W——, age 30 (?), was brought to the London Hospital hemiplegic on the right side, and unable to utter anything but "yes" and "no," and those words at first she used without precision. When asked to tell us where she lived, we thought she made abortive efforts do so as she moved her lips, but she uttered nothing. The patient had been paralysed two days, but how it came on we could not get to know. She was, all the time she was in the hospital, quite sensible; and except for her paralysis and loss of speech, she seemed very healthy. She could put out her tongue readily, and could eat and swallow well. There was never any albumen in the urine; there was no evidence present of syphilis, but from her associations she may very likely have had it.

Her pupils were not affected; they were neither too large nor too small. On using the ophthalmoscope, I found in the left eye, "The margin of the optic disc, especially the apparent outer margin, is indistinct and gradually smoothes off into the neighbouring fundus. The inner margin is also indistinct, but looks more ragged than smooth. The arteries are not apparently much diminished in size. The changes in the right eye are nearly the same."

The nurse of the ward remembered that this patient had been in the hospital about a year before for "fits," but remembered nothing more.

Two days later the patient tried to tell her age by her fingers, and this I several times afterwards asked her to do. It was nearly always about 30. When she wanted anything she could, the nurse believed, beckon for it. On April 11, she could say the word "better." This, and "yes" and "no," were the only words she could say. She could not say "betty" nor "batter," nor any other substitution—compound of the word. In trying to say

"betty," she said "bet" well; and although she seemed to make great efforts to produce "ty" as the next syllable, "ter" was always produced instead. Then she smiled and shook her head, as people do who are half amused and half vexed at failing in a little thing.

April 16. She had a convulsive attack. She was, it was said by the nurse, convulsed on both sides. She bit her tongue. She seemed, afterwards, to go on in pretty much the same condition.

May 30. Can say "yes," "no," "better," "bread," "pan," "net," and she made Dr. Parker understand "Monday." I urged her to say "Tuesday," she said "Bet-day;" "Wednesday," "Wetsday." It may be said that this was owing to defect of articulation. Very likely; but not to a difficulty of articulation, due to actual paralysis of the muscles of articulation. It is at least a mistake to suppose that in cases of this kind there is any precise order as regards linguals, dentals, &c. When asked to say "rub," she made a sound most like the attempt to pronounce "pf;" "egg," "et" and "ext;" "head," "et;" "end," "edta."

She did not improve in paralysis, but her power to make signs became gradually greater. On June 19, when asked how many days there were in the week, she said "stels," then held up six fingers and then seven.

This patient seemed to see well. Probably if she had learned to read, which is not likely in one of her class, she would have lost the power even of reading to herself, and of course she could not read aloud. The only way of getting any trustworthy information was by looking into her eyes.

Into the nature of the defect of speech I need not enter here. The "general faculty of language" was never probably quite lost. We meet with all degrees of defective power of expression, but in nearly every case there is the same great clinical association viz., hemiplegia on the right side. To this rule, I have myself seen but few exceptions; and I have never seen so great defect of speech, as this patient had, with hemiplegia on the left.

CASE XV.—*Amaurosis after Hemiplegia on the left—Defect of Speech with Hemiplegia on the right.*

I was consulted at the Hospital for Epilepsy and Paralysis by a patient who had defect of speech and paralysis on the right side. He was also amaurotic. The defect of speech came on three months before suddenly, and was no doubt due to disease of the left hemisphere near to and involving the left corpus striatum, for he was paralysed on the right side. His speech was only partially affected, and apparently it was only a certain difficulty of articulation which I have called "Ataxy of Articulation" (See remarks with Case XIV), and which I have found to be due to disease near to and involving the corpus striatum, and to be associated with difficulties of other acquired methods of expression, as in spelling, &c. The attack which produced such symptoms in a man 40 years of age, was doubtless due to apoplexy; and the fact I have here to mention, that he was insensible and "deranged" for three weeks after, points to a large clot.

He had atrophy of the optic nerves; the edge of the discs being indistinct, and the vessels small. I was interested to hear the patient's wife say that the defect of sight followed an attack of hemiplegia the patient had had five years before, and that attack had been on the left side.

The case excited great interest, but the patient only came once or twice; and I unfortunately knew nothing further of him than that he died, but in what way I could not learn.

CASE XVI.—*Pain in the Head and discharge from the Ear from Childhood — Epileptiform attacks with Epileptic Hemiplegia — Double Amaurosis—Autopsy.*

For the opportunity of seeing and of being present at the post-mortem on this case, I am indebted to Mr. Jabez Hogg. The following notes are from joint contributions by Mr. Hogg, Mr. Calthorpe, and by myself.

The following is by Mr. Hogg:—

A. B. H., æt. 34, public singer, admitted June 8th, 1863, with loss of sight in left eye and partial strabismus and diplopia.

He complained of having suffered from headache for some days, arising, as he thought, from over-work, having at the time to sing at three different public rooms every night. He was well known as an excellent comic singer, and was remarkable for the great energy he threw into the various characters. He described the first attack as coming on after having made greater exertion than usual, when he rushed off the stage into his dressing-room, and there remained, he believes, for a short time partially unconscious; but this could not have been for very long, for, after the administration of some stimulants, he recovered sufficiently to fulfil another engagement at some distance, but this only served to increase his languor and the pain in his head. The next day he saw his medical man, who prescribed for him, and assured him that it would all pass off. In a day or two he was no better, and it was then that he found the sight of his left eye so much impaired that he came at once to me.

There was a peculiar prominence of both eyes, producing a somewhat vacant stare. He complained of slight headache, but was otherwise quite well, and able to take his food with an appetite. The diplopia annoyed him very much and he walked with an unsteady, uncertain gait. He could scarcely tell me whether he had sight in his left eye, and upon testing him I found he could just distinguish daylight. The right was not very much better, but with it he could read large print, but not the letters on the houses on the opposite side of the street. The pupils were nearly fully dilated.

[When I some time afterwards examined the optic discs, they were in the condition of atrophy, as I thought after neuritis. I have no notes of the examination. It was afterwards ascertained from his wife that the attack described by Mr. Hogg was not the first. One day, shortly before he was going to wash himself preparatory to going out to sing, he said, "Tim, I feel so giddy, I have got such a strange sensation, I can't see, and I can't recollect anything." He had a trembling—not an attack of shivering—but did not become insensible. In a quarter of an hour he dressed himself, and was going out to sing, when, at the door, "he felt and fumbled about." He then went to bed and was sick, but he had no convulsive attack—J. H. J.]

He was treated; but on July 7th he left the hospital not in the least relieved, the sight, if anything, worse than it was before.

He continued an out-patient for some time, but at last became totally blind.

October, 12, 1863. His wife came to me begging I would go and see him at his lodging in the Borough. I did so, and found him in an epileptic fit; the left leg and arm paralysed (hemiplegia); but the moment the fit subsided he recognised my voice. I prescribed for him, and subsequently saw him many times. The fits gradually subsided, but he remained very feeble, and with the left side partially paralysed. He, however, recovered, and had no return of the fits for some time, during which I recommended him to place himself under the care of Dr. Hughlings Jackson, at the hospital in Queen Square.

The following is by Dr. Jackson:—

I saw the patient in November, 1863. He had then had another fit, and had slight hemiplegia on the left side.

January 17, 1864. I was asked to see him at home. I was told that he had had twenty-five fits that day. I was told also that they affected both sides, but in two that I saw the left arm and leg and both sides of the trunk were affected. Both eyes were turned to the left, the pupils were quite equal and did not alter in size during, before or after a fit. After each of these fits he vomited a quantity of dirty green fluid, and had done so once before. When the convulsions passed off he never became fairly conscious, but moaned. He put up his right hand to his face, but never moved the left spontaneously. When it was pinched, however, he moved it slightly and moaned more loudly.

I did not see much more of him at this period, as he was then under the care of Mr. Woolley, who took charge of the case.

Next come two notes by Mr. Hogg:—

April 27, 1864. I saw H. who had been entirely free from fits and had once sung for a benefit.

March, 1865. H. called to see me, looking remarkably well, and telling me he had had no return of fits since I saw him in April, 1864. N.B.—Not the smallest change in sight.

Soon after this note the patient began to be ill again.

For the following interesting notes I am indebted to Mr. Calthorpe:—

About May last I first saw Mr. H. He was then suffering from a series of epileptic fits, of the same character as those he had experienced before. He had had, I believe, when I first saw him.

about 100 fits, chiefly at intervals of a quarter of an hour. I did not see him actually in a fit, but was informed, that during the intervals he was possessed of all his faculties (save sight), and only complained of slight pain in his head. There was no paralysis or loss of sensation, and but very slight ptosis of the left eyelid. The whole number of fits in this attack was about 250.

Within the last two or three months he had two other slighter attacks; in each of which he had forty or fifty fits. But as his friends did not send for assistance, I have no notes whatever of the seizures. I was told though, that they differed in no way from the previous ones.

On the 7th of October, 1865, I attended again for Mr. Hogg. He had had nearly 200 fits since the Friday (the 6th October) previous. I saw him in a fit, which commenced with a rather hurried inspiration, followed by a rapid rolling of the eyes (widely opened) from side to side; then ensued violent convulsions, principally affecting the left arm and especially the hand. The fit lasted, as usual, about a minute, and the instant the convulsions were over, he was sensible of external objects, and replied readily and aptly to any question put to him. When asked where he felt most pain, he raised his right hand and put it over his right temple. The left side of the face looked paralysed; there was slight ptosis, and the left leg could be but slightly moved.

On Monday, the 9th, I saw him again: the number of fits had increased to nearly 450. I saw him in two fits. They were accompanied by more determined ptosis of the left eyelid, though still not to any great extent. The left arm was partially paralysed. If lifted, it fell with a bang, but could be shifted, by a seemingly great though voluntary effort, to a more comfortable position. The left leg was completely motionless. He was still perfectly sensible between the fits, but evidently fast sinking; indeed I was told that the last twenty or thirty fits had not been so powerful as usual. The pulse was small and slow. He had about fifty more fits during the night, making in all nearly 500, and he died on Tuesday morning, about 5·0 a.m. The fits ceased about two hours before death, and he died perfectly quietly, so much so, indeed, that the exact moment of death was not noticed. The duration of the last attack was about ninety-six hours.

It is somewhat curious that on the Thursday night he went to bed, saying that he felt better than he had done for a long time.

previously, and his wife was awoke some time during the night by his being suddenly taken with a fit.

The following is by Dr. Jackson :—

The autopsy was made by Mr. Calthorpe Mr. Travers, and myself. It was quite certain that we should find disease of the right hemisphere. On removing the calvaria, the convolutions at the side of the hinder part of the anterior lobe on the right side seemed to be atrophied, and there was here more fluid than on the other side, and the brain seemed shrunken. On further examination, it was not easy to make out the exact number of convolutions affected. Indeed, on making a section, several of them seemed to be little more than bags of semi-fluid matter. On comparing it with a wood-cut from Dr. Arbutin's work—"Considérations sur les Localisations Cérébrales et Particulier sur le Siége de la Faculté du Langage Articulé, 1863."—I made out that the disease was chiefly in front of the furrow of Rolando, and that it affected at least the hinder two-thirds of the 3rd and 2nd frontal convolutions. The brain here was nearly fluid, and soon gave way into a cavity which extended inwards and involved the corpus striatum slightly. There was another such cavity in the hemisphere just above the ventricle and near the superior longitudinal fissure. The rest of the brain was healthy. By the microscope I could find nothing except fat-globules and granules.

I have to regret that I did not ask Mr. Calthorpe to examine the left petrous bone. I had forgotten then that the patient's wife had known that for thirteen years he had had an offensive discharge from the left ear, and his mother had said this had existed from childhood.

Now, it seems, too, that he had always "suffered from his head," even from childhood. His wife said that before the events recorded (*i.e.*, before 1863) he used to be liable to be laid up with "nothing but pain in the head," and that he used then to vomit. The vomit was sometimes clear, and sometimes yellow. The doctors used to treat him for it with iodide of potassium. The pain was said to be neuralgic, but it had never affected his face. It was all over his head.

CASE XVII.—*Headache, Amaurosis, Epileptiform attacks and Epileptic Hemiplegia.*

Sarah S., æt. 21, a well-built, good-looking woman, came to me for epilepsy at the Hospital for Epilepsy and Paralysis, April, 1863. She was then also quite blind. The following was her history:—

Twelve months before I saw her, in the early part of 1862, she began to suffer from severe pain in the head. She had had one child twelve months before, but it died. She had not been regular for some time before the pain began. The pain passed from temple to temple, and was all in front of the ear. She never had any pain at the back of the head. It was slight at first, and then became very bad. It rarely went off, only just when she dozed at night, but she got no continuous sleep. It was bad for three months, and then ceased, but when it had ceased her sight began to fail. At first it failed at night only; she could walk well in the day, but could not at night. "There seemed to be fog before the eyes." The left eye failed before the other. In one month sight was quite lost, and she has been blind ever since. It is worthy of remark, that she had no vomiting at any stage of her illness.

Nearly four months before I saw her, *i.e.*, eight months from the first, she began to suffer from the fits for which she consulted me. She was then, she said, except for the blindness, in perfect health, and she has always been so since she has been under my care.

One day, when swallowing a piece of apple, she felt "something jaw-locking her," and her mouth was drawn to the left side. She then fell back, and her left arm and leg were convulsed. She was sensible throughout, and has been in most of the others. She bites her tongue, and generally on the right (*sic*) side. She can talk after the fit, but usually sleeps, but only for a few minutes. Next day she feels ill, and has a headache. Since the first, she has always known that the fits are coming on by a feeling of "numbness" in the left arm and leg, left side of the face, and left side of the tongue. She has pain in the arm and leg, and the hand "draws up" and "claws." I never saw her in a fit, but one day I saw her at the onset of one.

I saw that the whole left side of the face was twitched (orbicularis palpebrarum included), but being urgently required in another part of the hospital, I could not wait to see the rest.

When I see her at the hospital she looks perfectly healthy, except that she is blind; but she has once or twice complained of feeling weak on the left side after the fits. On one occasion she said she lost the use of the left arm and leg for a quarter of an hour; slight epileptic hemiplegia. Once too, but once only, she said that she had pain, a "nasty numbing feeling" up the right arm. Recently also, as is common in epilepsy, she has had occasional spasm in part only of the muscles usually affected in the fits, viz., in the left arm.

She has had two or three fits in a week, *i.e.*, about eight before she saw me.

Practically, she is quite blind, but can just perceive the light dimly with the right eye. The discs are quite white and look flat, their margins are indistinct, and round about them the fundus is darker, as if irregularly pigmented. Here and there on each side is, as it were, a process or stream of this colour running out into the healthy fundus, the rest of which is normal. The vessels do not seem to be much diminished in size. The pupils are large. There is no exophthalmos.

Her sense, of smell and hearing are quite good, and her speech is perfect in every sense. She seems to be very intelligent. In short, the whole of her physiological symptoms are double amaurosis and hemiplegic left epilepsy. The hemiplegia points to disease of the right side of the brain affecting—probably secondarily—the motor tract; but what that disease is there is no evidence to show.

Case XVIII.—*Extensive injury to the right hemisphere—Double amaurosis—Hemiplegia, and epileptiform attacks.*

This patient was under Mr. Paget's care in St. Bartholomew's Hospital. He permitted me to see the patient, and was good enough to allow me to take him under my care for epileptiform attacks. I need scarcely point out that this is another case which is full of points of interest. In the first place, it seems likely, as I believe Mr. Paget thought, that the case had been one of cerebral abscess. Then the great intelligence of the patient is a point

worthy of great attention. He speaks quite well, and has written me a clear and able account of his case. It is interesting, too, as a case of paralysis with epileptiform convulsions. Again, there is an aura, *i.e.*, the convulsion begins very locally, in the hand, and this can be stopped by the ligature. It may seem remarkable that a ligature should stop the fit, when clearly the disease is organic, but it is, I think, frequently so in epileptiform seizures from disease of the hemisphere. Then this patient has partial fits, as he calls them. This case resembles Case XX.* in the physiological symptoms. In both, the same part of the brain is, I believe, damaged; although, in one, from an injury, in another by syphilitic disease of the connective tissue. It is the same region as was affected in Cases XIII., XVIII., and XIX. That the fits are kept off by the bromide of potassium is a point of extreme interest, not only as regards the empirical fact, but as showing that in epilepsy we have not only to treat the original local disease, which produces the epilepsy, but some secondary condition to which the local disease gives rise.

A man, now 23 years of age, was struck on the head, Dec. 3, 1863, by a shovel. The immediate result was a scalp wound, for which he was kept in a hospital in New York fourteen days, and was then discharged apparently well. A week later he began to be giddy, rambled about, and talked nonsense, and was supposed to be going out of his mind. His head swelled, and he was told at the hospital that he had a "puffy tumour" on the right side of his head. He became comatose; and on January 3 was trephined. He was afterwards told that this was done on the supposition that there was an abscess "pressing on the brain," and that at the operation "four or five ounces of matter were let out." He did not recover his senses, he says, until March 4th.

[Here, out of order, I may state to have done with the description of the organic disease, that there is now wanting a large tract of bone on the right side of the skull (4in. by 4), part of the frontal, part of the occipital, and nearly all the parietal bone.]

The first fit, or at least the first of which he knows anything, occurred on March 14th—101 days after the injury, 80 days after the trephining, and 10 days after recovering consciousness.

* This case will be related in the next number of this Journal.

The fit began in the fingers of the left hand, and it gradually ran up the arm, missed the neck, and affected the face. The face was drawn. After this he became insensible, and found, on recovery, that he had bitten his tongue on the left side. He was told that the fit only lasted two minutes; he did not sleep after it, and in half an hour felt well, except that the left arm and leg were weak, and they have been so ever since. He had in fourteen days a series of five fits, and for some time four or five at a time, about every fourteen days. At the end of about five such periods, he had no more until an operation on August 20. The bone came away on August 3, and on the 20th an unsuccessful attempt was made to bring the skin over the gap. He then had three fits. Since they have been much less frequent.

They all begin in the same way, viz., in the hand. The arm is now decidedly paralysed, so that he cannot use it for any purpose. The leg is weaker than the other, but only slightly so.

This patient has atrophy of each optic disc; but they are now pretty well margined, but there is a slight irregularity at their edge, which makes me conclude that the condition of atrophy has followed neuritis. It is the condition into which neuritis ultimately passes. The vessels are not very much diminished in size. There is no loss of smell, and the patient is very intelligent. So long as he takes the bromide so long is he free from fits.

Case XIX.—*Hemiplegia on the left side with Amaurosis—Autopsy.*

Thos. R——, age 50, came under my care in July last. He was transferred to me by my colleague, Dr. Parker.

The patient had been hemiplegic on the left side three months. It was believed to have begun suddenly, but he was never able to give a clear account of it, and his wife, when she came to the hospital, was not in a condition to be relied on. The patient lay in bed, and was never fairly alert, but was not actually insensible until shortly before his death. He could always talk properly; and on Sept. 4, he said his sight was bad. Mr. F. M. Mackenzie, Dr. Woodman, and myself examined the eyes, and found that he had optic neuritis on each side. He used

to have great pain in his head, and this was his chief complaint; but he had not had any vomiting.

He died Oct. 26. The right hemisphere bulged laterally and on slicing it, the knife went through a dirty yellow diseased mass which was just under the thinned grey matter of the convolutions. This was nearly on a level with the lateral ventricle. The corpus striatum seemed to be intact in its ventricular portion, but on cutting through it, it was found to be involved in the mass. The mass was about the size of four walnuts, and extended up to and so as to render prominent the grey matter of several convolutions which were soft. These included the hinder part of the third right frontal. The convolutions of the island of Reil were hardened and looked corrugated.

The mass was of a dirty yellow colour, and was made up of nothing whatever but granules. There was no fibre, and there were no blood crystals. This was the only part of the body diseased.

Sequel to Case II, p. 398.

This boy is dead. He was eight years old. As my last paper was passing through the press the boy had begun to get worse again, and on April 3rd I made a note to that effect. Shortly before that date his friends had considered him well, except for his blindness, and of the extent of it they were not fully aware until Dr. Septimus Swyer and myself pointed it out to them.

The boy gradually grew worse, the pain becoming more and more severe, and the vomiting returning.

I saw him only at intervals. On July 20th I have a note that he complained that the pain was chiefly intense in the right ear. On the 15th of August I saw him again. He was then dull and drowsy, and yet, although peevish, not obviously unintelligent. For three weeks he had weakness of the left arm. He could move it, but with much tremor, and it was of but little use. As he was lying in bed, and was disinclined to be disturbed, it was not easy to demonstrate that the leg was weak, but doubtless it was so. There was now also swelling of the side of the head and face, pretty much as there was at the autopsy. He died Nov. 5th.

Post-mortem examination by Dr. Swyer, Dr. Septimus Swyer, and Dr. Hughlings Jackson.

Nov. 8, 1865. The right side of the head was more rounded

at its fore part than the left. This was chiefly due to swelling in the temporal region. The swelling extended to the region of the right superior maxillary bone and thence to the inferior maxillary. Both these bones were believed to be involved, but we were not permitted to examine them as much as was desirable. On looking into the mouth we saw a ragged dirty-looking tumour growing from the right side of the fore part of the palate.

On sawing off the skull-cap, the sensation was like that felt on sawing off the skull-cap of a much younger child. The saw-cut in the right temporal region was soon filled with a dirty bluish-coloured matter. The skull-cap came off easily. It was thinner than usual, although the cranial cavity had not much enlarged. The brain was everywhere easily detached from its bed, except at the side of the sella turcica, and at the inner end and front aspect of the petrous bone. From these parts the brain was cut away, and pieces of the tumour adhered to them.

It was then seen, that so far as the base of the skull was concerned, the disease was limited to the middle fossa. This was perforated, and through the hole the finger could be easily passed into the mouth, the channel being through the tumour seen in the mouth.

The disease of the brain was a tumour, involving the middle lobe. A great part of the lobe was in a sloughy condition.

No doubt had been felt by any of us for some time before death that the disease was malignant. The autopsy showed that it was. The tumour (at least the part of it adherent to the bone) was soft, and exuded much juice on scraping.

We found also secondary deposits in the lungs. At first the feel of the lungs made us think of miliary tubercle, but they were too soft. Then there were larger masses on the surface, which looked cartilaginous, but these, like the smaller ones, were soft, and yielded much juice on scraping.

The tumour from the brain, and the large and small masses in the lungs, showed many nucleated oval cells (or nuclei), all of them containing one, and some two, or more nuclei (or nucleoli). The juice scraped from any of those parts showed crowds of such cells or nuclei.

Probably the tumour began in the bone or the membranes, but this is by no means certain.

(To be Continued).

A LECTURE ON POSTERIOR STAPHYLOMA, WITH SPECIAL REFERENCE TO TWO SINGULAR CASES.

(*Moorfields, June 2nd*, 1865.)

By J. F. Streatfeild.

In the last edition of our most comprehensive English work on eye diseases, published in 1854, we read of staphyloma posticum, that it "has been met with on dissection," and nothing more is said of it. That there was such a disease, or, even more, that it produced such results as are now well understood by posterior staphyloma, was hardly known before the invention of the ophthalmoscope. The title is not a very good one. Posterior staphyloma (or "sclerectasia posterior") is rather a name for the effects of disease than for the disease itself; but, until we know more of the mode or modes of production of posterior staphyloma, it is convenient, and, I think, speaking generally, it is better than the alternative of "sclerotico choroiditis posterior," which may or may not have existed, certainly does not always exist, in cases such as those of which I am about to speak. But then, again, in posterior staphyloma, so called, it must not be understood that there is always a staphylomatous part that is so only, or even particularly, to the extent of the disease, as it is seen with the ophthalmoscope, but *there may be only a general staphylomatous extension of the coats of the eye posteriorly.* Posterior staphyloma might be described in different ways of classification, but I propose to consider it under two heads—the stationary and the progressive staphylomata. From whichever kind the patient suffers he probably comes to you complaining of myopia. In the latter form he probably complains of increasing myopia, and other and more serious complications may exist, or will be likely to arise. In the stationary kind of posterior staphyloma the great characteristic of an ophthalmoscopic examination is a white crescentic mark touch-

ing the optic nerve entrance, and embracing it more or less, that is to say, its concavity corresponds with and is joined to some part of the circumference of the optic nerve entrance. Not unfrequently, however, it is not thus partial in its extent in relation to the optic nerve. The horns of the crescent may encircle the nerve altogether. If this white mark completely surrounds the nerve it is not equally broad, but, as in the case of the simple crescent, its greatest breadth is towards the posterior pole of the eye, that is to say, to the *outer* side of the nerve. Sometimes it is below the optic nerve entrance, and but very rarely in other positions. Liebreich says "I have never seen it above" the optic nerve entrance, but in our last course of ophthalmoscopic demonstrations we have seen a case in which there was a small crescent in that position. Some of you will, no doubt, recollect the case. The outer margin of the crescentic mark generally forms a tolerably regular curve, and when it extends nearly or quite around the optic nerve entrance it might be described as a part of the circumference of a circle, somewhat larger than the optic nerve entrance, and of which the centre is to one side (generally external) to that of the nerve. The convexity of the crescentic mark is generally more or less bounded by streaks or by a continuous line of brown pigment. Beyond the dark margin is the normally bright red choroid, with its epithelium, etc., all apparently unchanged, and the contrast between this and the white of that portion of the fundus to which the disease appears to have been limited is very striking, especially as it is of a peculiarly glistening white, and the limits of the two are defined by the dark pigment of which I have spoken.

But in the progressive form of posterior staphyloma the choroid in the neighbourhood of the white mark generally is paler than elsewhere, or it is so in parts, and the colour may be abruptly margined in gradations, or it may be shaded off from the staphylomatous part, or, more accurately, I should say, from the white space. The limits of

the white space become irregular, and instead of one even curve, its outline is of several bows jutting out, with angles towards the optic nerve entrance. The retinal vessels crossing the staphyloma take a straight course, as if stretched, and, by contrast with the light back-ground, they appear very conspicuously. In some cases irregular patches of choroidal pigment are seen in the white space and in the thinned parts in the neighbourhood of it which are threatened by its advance, and generally the optic nerve entrance appears oval, or flattened on the side towards the staphyloma. This is because the optic nerve entrance, or a part of it, has been drawn to one side and backwards in the bulging of the sclerotic, so that the disc is seen in profile, or partly so; and here it is worth while to observe that the long diameter of this oval is at right angles to the direction of the greatest extent of the staphyloma.

Cases of posterior staphyloma are very easy of ophthalmoscopic examination; indeed all cases are so in which the fundus is easily lit up, the media being clear, and in posterior staphyloma there is an abnormal proportion of light reflecting surface in the back-ground of the eye. The eyes of fair complexioned people, and all those in which the choroid is paler, having less pigment than usual, are, for this reason, most easily examined.

To those who are unused to ophthalmoscopic examination it may be useful here to point out to you the differences that may be observed between posterior staphyloma and some of the other cases of ophthalmoscopic disease. Among the cases which I have known to be mistaken for posterior staphyloma are those of results of inflammation of the optic nerve, or the "white atrophy," of which we see so many examples. But in these cases the white optic nerve entrance is not larger than the usual healthy optic nerve entrance, but probably smaller, whereas posterior staphyloma is an addition to the size of the optic nerve entrance, and the staphyloma (including the optic

nerve entrance) considered as a light spot in the red choroid of the fundus, is more or less larger than the usual size of the optic nerve entrance, as the staphyloma is larger. Then, in a case of posterior staphyloma, the optic nerve entrance is to be seen as a pinkish round or oval spot in the white space. It can be found at once by observing the part to and from which the retinal vessels go. Also, in posterior staphyloma, the arteries and veins are normal in size, and relative proportions, and are very distinctly seen, all of which is contrary to that which is generally seen in a case of "white atrophy." In glaucoma there is a whitish margin to the optic nerve entrance, but it is dull as compared with posterior staphyloma, and there is no one part at which it is much wider than another. Then, in glaucoma, there is the visible pulsation of the vessels of the optic nerve entrance, and they are bent in their course over its margin, instead of being very straight, as in crossing the staphyloma. In glaucoma, too, the patient has been getting more and more far-sighted, instead of short-sighted, as I have said of posterior staphyloma. Besides the ophthalmoscopic distinctions there are many well known means of diagnosis of glaucoma, into an account of which I need not now enter. Glaucoma may be superadded to posterior staphyloma. Glaucoma or "white atrophy," especially the latter, could hardly be mistaken for any but the stationary form of posterior staphyloma. But some cases of inflammatory effusions and changes in or near the optic nerve entrance more resemble the larger posterior staphylomata. If of the choroid, it is probably less exactly applied to the optic nerve entrance than posterior staphyloma is, and it is not so glistening in appearance. If of the retina, around the optic nerve entrance, as in some cases of Bright's disease, there is an indistinctness or a complete obliteration of the outline of the optic nerve entrance, whereas in posterior staphyloma the optic nerve entrance is easily distinguished in or by the side of the white space. Moreover, in these cases there is not the

same tendency to form definite outlines of arches outwards from the optic nerve entrance. In these retinal cases also there is not the same brilliancy of reflexion that characterizes a posterior staphyloma. And it is to be remembered that if the light coloured space, which is identified with posterior staphyloma, is not always quite white, it is very bright looking, never of a woolly white or of a "dead" colour. If, in eyes otherwise diseased or in old people, the media are not clear and colourless, the fundus, and especially the white exposed sclerotic has often a more or less yellow tint.

The disease is called "sclerotico-choroiditis" or "staphyloma," the result of the former; but, besides some slight hyperemia in parts of the choroid near the optic nerve entrance or the white space already existing, or in the thinned parts, which is sometimes to be noticed, we see very little appearance of inflammation, on an ophthalmoscopic examination. In some cases of stationary staphyloma there is no appearance of localized bulging of the coats of the eye, but in some of the advanced cases it is very evident. The retinal vessels crossing the staphyloma appear to be stretched, or on a different level to those of other parts of the retina. And the optic nerve entrance is apparently oval, or drawn away to the white or light coloured space, where it is most extensive. Besides, it has been found, in these cases, *on dissection*, to be bulging or staphylomatous. The choroid and retina recede with the sclerotic. The choroid becomes stretched and then atrophied. The retina is so also, or it may even become detached from the choroid. The choroid being atrophied, its vessels obliterated, and its pigment gone or heaped up irregularly in some places, the white sclerotic is exposed, and reflects the light brilliantly—"the white of the eye," it is in fact. Perhaps in slight cases of posterior staphyloma the choroid is at first only separated from the margin of the entrance of the optic nerve to some extent of its circumference; but in other cases we see the choroid gradually

disappearing altogether, thinning progressively around the optic nerve entrance, and from it especially towards the posterior pole of the eye, so that the yellow spot is likely to become involved in all cases. The inflammation is localised in that part of the eye upon which, especially if there is an intraocular hypersecretion, pressure would be likely to take effect, and if it is principally and in the first place a sclerotic affection, that there is so little evidence of it ophthalmoscopically, may be explained by its being then covered by the choroid coat, which becomes stretched and atrophied when the sclerotic is first weakened by the inflammation, and then bulged outwards.

That the antero-posterior axis of the eye becomes thus lengthened will of course account for the myopia.

Sometimes posterior staphyloma seems to be a congenital affection. I have now a little boy attending the hospital, who was first brought to me as an infant for this disease. His eyes are always rolling laterally.

In advanced cases, besides the myopia as a result of the advancing disease, the patients become more and more amblyopic, as in one of the cases I am about to relate to you.

The cases are two; both were attending our last evening course, and I have made careful drawings of both cases, which I have brought to show you. Both are instances of the progressive form of the disease, and both of them are in some respects very remarkable, hardly alluded to in any of our books describing ophthalmoscopic diseases, and not figured in any of them. The cases are also quite different.

The first is—

Cornelius Cronin, who came to me here on the 6th of February, in the present year. He came from Cork, where he had been educated for a schoolmaster. For eight or nine years, he says, he had studied. He was very fond of it, and had "strained" his eyes. He had learnt several books of Euclid, as he informed me. For some years past his imperfect vision had obliged him to work as a labourer,

and, seeking work, he had come to London. He had evidently read more than most labouring men, and was much better educated in every way than they generally are. He told me he had "no permanent residence, but had been recently dwelling in St. Giles!" He had never sought any advice, but his sight being so very defective, and because he squinted, his friends sent him here. He was 25 years of age, slight and fair, in good health. His parents were not short-sighted, nor had they any defects of vision, but they had not had the education he had had. When he was 14 years old his sight began to fail, and to get shorter and shorter. In three or four years afterwards he had to give up reading and writing. The external strabismus of the right eye began about this time; it was of about three lines when I measured its extent. Since that time his sight has been gradually getting worse and worse. With the right eye he could hardly spell No. 16 of Jäger's types, at any distance or in any direction. With the other he could, at six inches distance at farthest, read No. 1 fluently. So that I suppose he meant by his failing sight that it was so gradually in the right eye tested separately, and that with the left eye he could only, as at present, see objects brought nearer and nearer to the eye. As a boy he had kept sheep, and he could make them out at a distance like any one else. He had sometimes seen muscæ, like strings of pearls, when looking at a light. He had seen no flashes of light; he had felt no pain or any sensation of tension in the eyes, no confusion or distortion of objects. He had never worn glasses. He had never had inflamed eyes, or received a blow on either eye. At some times his lids used to be agglutinated in the morning. His general health was very good, and he had had no illness. But he complained that if he stretched himself, that is, if he extended his arms over his head, a mist used to appear before him for an instant, and that when he looked down and then up at the light he saw something like gauze descend from above and his left, to below and to his right side (over the

left eye). In order to see best, he used to shut the right eye altogether and the left partially. There was a constant tremulous motion of the right eye laterally, bringing it each time a little way inwards. The movements were not quite regular; they were about 50 in the minute. The tension of either eye was normal, certainly not abnormally great. The length of either eye from before backwards seemed to be very great when converged.

When I examined his eyes with the ophthalmoscope I at once saw the appearances depicted in the plate. The singularity of the case is summed up in this—*a posterior staphyloma existed without any connection with the optic nerve entrance.* An ordinary posterior staphyloma existed in this eye, the right, but chiefly on the inner side of the optic nerve entrance, away from the extraordinary staphyloma which was quite separate and altogether unconnected with it. This the ordinary staphyloma was not therefore in the most usual situation, or rather, as it extended round the whole circumference of the optic nerve entrance, it should be said that the crescent, the widest part of the staphyloma was rather, but not exactly, on the opposite side of the optic nerve entrance to that on which it is usually found. As far as the ordinary staphyloma was concerned, the left eye of the patient was symmetrically affected, but there was no other resemblance between the two eyes, and no initial stage of the extraordinary staphyloma, that could be recognized, in the left eye. The larger and extraordinary staphyloma of the right eye was of about twice the diameter of that encircling the optic nerve entrance. It occupied the region of the yellow spot. It reflected light very brilliantly as from the bottom of a hollow, from one part or another particularly as the light was thrown into it. Its margin was surrounded by irregular streaks of black pigment, some of them being within the margin, as if it had increased in size, and they had been left there. Its margin fell a little out of the circle on the side towards the optic nerve entrance, as if it would be likely to join with the

Inverted Image_Right Eye.

Plate I.

J. Erxleben, lith

J.F. Streatfeild, del.

Hanhart, Imp.t

staphyloma in that situation; but there was a broad bridge of perfectly healthy looking choroid, retina, &c., between the two staphylomata. Many of the short ciliary arteries were seen entering the large staphyloma, through the sclerotic, generally in pairs, through little slit-shaped openings common to the two. Some of them seemed to me to go to the sclerotic itself. Their course seemed also to indicate the deep hollow of the staphyloma. They were very fine, and did not seem to have a forward course, but were generally curved into the staphyloma. The choroidal epithelium in the neighbourhood of the large posterior staphyloma was all around it much more distinctly pigmented than elsewhere, and on the inner side of the optic nerve entrance the choroidal vessels could be very distinctly seen.

[To be continued.]

EXPLANATION OF PLATE.

The coloured plate shows the inverted image of the right eye of Cornelius Cronin, and exhibits a large ordinary staphyloma surrounding the optic nerve, and a large, almost circular, extraordinary one occupying the position of the yellow spot. The details are sufficiently described at page 86.

THREE CASES OF CANCER WITHIN THE EYE-BALL IN WHICH SYMPTOMS OF ACUTE GLAUCOMA WERE SUDDENLY DEVELOPED.

By Jonathan Hutchinson, F.R.C.S.

The three cases which I am about to relate illustrate some important points in the pathology and diagnosis, both of detached retina and of glaucoma. Two of them also convey an important practical hint.

Case I.—About four years ago a woman, aged 45, came to the Hospital in the third day of what appeared to be acute glaucoma. The pain in her eye had set in rather suddenly, and it had been most severe. Her sight had been failing for several months. She had had photopsiæ muscæ, &c., but there had been no pain until the present attack. The globe was dusky and congested. The cornea was steamy, and the media returned a dull, reddish appearance, as if stained with blood. The tension of the globe was much increased; and in the belief that I had to do with a case of acute glaucoma, I advised an immediate iridectomy. This was done, and great relief to the pain was afforded. She, however, regained no sight, and the eye a month or two later again became very irritable. As the media were still opaque, and the organ clearly lost, I now advised extirpation. She did not submit to this operation till some weeks later. After its performance I found a growth of melanosis between the choroid and retina, close to the optic nerve. It had perforated the sclerotic, and a small growth, the size of half a hazel nut, bulged into the cellular tissue of the orbit. After the operation all went on well. At the end of a year, as she had not presented herself for examination, I wrote and requested her to do so. At this time she believed herself quite well, but on examining her orbit I found beneath the conjunctiva numerous little black dots of melanosis. On cutting these away more came into view, scattered amongst the cellular tissue and fat. It was necessary to clear away almost all the contents of the orbit. Two months later she had large quantities of blood pass by stool, and on ex-

amining her abdomen I found her liver very distinctly nodulated. She had by this time become cachetic. I have not seen her since, and the probability is that she did not live long after the last note.

I mention this case on account of the occurrence of an attack closely resembling glaucoma in connection with malignant disease of the choroid. In the next two cases the pseudo-glaucomatous attack was yet more acute and severe.

Case II.—John R., a healthy-looking man, æt. 40, was sent to me by my friend Mr. Steele, of Reigate, on account of failure of sight of the left eye. He said that for the last six weeks there had been a fog before the eye; that he had seen "silver stars," "black specks," and a "waving of the air, like the waving of the sea in sunshine." His other eye was perfect, but with the defective one he could only make out letters of No. 20, and these he could only make out when held in certain positions. In the direct axis of vision he could see nothing. Lines of print looked, he said, zig-zag. These subjective symptoms seemed to point pretty clearly to detachment of the retina, and, on examination with the ophthalmoscope, this conjecture was confirmed. The detachment was in the upper part, and extended to the margin of the optic disc. It was easily seen, but the floating membrane presented no features which excited my suspicion. Several other ophthalmoscopists saw the case, and examined the eye. No one suggested the existence of malignant disease. There had been no pain in the eye whatever, and there was no history of injury. There was a history that four years ago he had suffered from a severe illness, which he was told was inflammation of the membranes of the brain, and for which he was salivated. As he recovered from it he found that his left arm and leg were weak, and they remained so for several months. Previous to this attack he had been liable to most severe headaches, but from these he had been since almost wholly free.

Believing the case to be one of simply detached retina, I did not advise any treatment, and he returned to his home in the country. This was in June, 1864. I did not see anything more of him until September, 1865, when he came to me with all the symptoms of the most acute glaucoma in his lost eye. The cornea was steamy; the tension of the globe much increased, and the sclerotic dusky. The pupil was large and fixed; but

with the ophthalmoscope I could obtain only a general dull reflex. He stated that the present symptoms had set in quite suddenly a week ago. He described the pain as having been horrible, and without intermission, and said that he had not slept since it commenced. The eye had remained exactly as I had seen it a year ago until this attack commenced. Had he came to me without my knowing anything of the earlier facts of his case, I should have been inclined to treat the disease as acute glaucoma, and perform iridectomy, but as he and I both knew that for a year past the organ had been lost as regards sight, I advised the radical measure of extirpation. To this he at once consented, and I immediately performed it. The eyeball, when removed, presented nothing unusual externally, but on looking at the section of the optic nerve itself I observed a small black spot. Having scraped this spot, and placed the result under the microscope, nucleated cells, loaded with pigment exactly like those of melanosis, were seen. On cutting open the eyeball a rounded growth of melanosis, the size of half a cherry, was found just above the optic disc, between the choroid and retina. It involved the optic disc itself. It was clear, from the condition of the optic nerve where cut across, that the portion left behind was also diseased. I, therefore, placed the man again under chloroform, and with some difficulty succeeded in excising a further portion of the nerve trunk half an inch in length. On examining this portion I found that it was quite healthy where cut across, and that the melanosis extended only about a line in depth from its distal end. This patient is at present in excellent health. It is about three months since the operation.

Case III.—Mrs. G., æt. 46, a dark complexioned woman, in good health, was sent to me by Dr. Hooper May, of Tottenham, in the beginning of last May. Six weeks previously she found that she could not see a candle when the left eye was covered. She had had but very trifling pain in the eye, and had never noticed muscæ, &c. She had discovered that the eye was blind almost by accident. The pupil dilated widely when that of the other eye was covered, and it remained quite fixed until the other was again exposed. On using the ophthalmoscope we found the retina extensively detached in the upper part. As in the preceding case, I noticed nothing which suggested the existence of any new growth. No treatment was advised, and for

several months I did not again see the patient. On October 13th she again came under my care. For three days she had been suffering very severe pain in the eye. It had kept her awake all night, and was still increasing in severity. The pupil was widely dilated, and fixed. The sclerotic was congested, and the media were hazy. The tension of the globe was very much increased. The cornea was steamy, but only slightly so. On examination with the ophthalmoscope I obtained only a dull grey reflex, and could not distinguish any retinal vessels. She willingly consented to the immediate removal of the globe. After the operation, bearing in mind the lesson learnt in the preceding case, I immediately cut the eyeball open. We again found a growth of cancer. It occupied almost exactly the same position as in the other case, but it was destitute of pigment—medullary, not melanotic. On looking at the optic nerve where cut across I found that the line of section passed through a portion of cancerous deposit. I therefore at once removed another half inch of the nerve.

The patient is at present in good health, but it is only about two months since the operation.

Remarks.—These cases well illustrate an occasional cause of detached retina, and the difficulties of making a correct diagnosis. The two latter cases were both of them carefully examined with the ophthalmoscope at an early stage, but only the fact of detachment was discovered. They prove also that new growths in the eyeball may become the cause of sudden attacks of intense pain, attended with increased tension, and all the phenomena of the glaucomatous state. *A priori* one would have supposed that intra-ocular cancer would be attended by gradually developed symptoms—slowly increasing pain, &c., rather than by a sudden outbreak.

A practical lesson suggested by my cases is the propriety of always examining an excised eye-ball on the spot immediately after its removal.

It is very annoying to a patient to be obliged to subject him to another operation within a few days of the first. Yet in both these cases it would clearly have been my duty to have proposed this measure had I not on the spot discovered, on opening the globe, that it contained cancer. In both the optic nerve was diseased at the point of section.

Note on the Excision of portions of the Optic-nerve-trunk.—In excising an eyeball the optic nerve is usually cut through close to the sclerotic. Should it be found afterwards that the part left behind is diseased, the excision of another portion is by no means an easy operation. It is buried deep in the wound amongst tissues infiltrated with blood-clot, and enough of oozing continues to occur to prevent the possibility of exposing it to view. According to my experience the only feasible way is to proceed by touch instead of sight. Passing the fore-finger deeply into the orbit, the nerve trunk can, with a little care, be discovered. A pair of strongly toothed forceps may then be guided down and made to seize the nerve trunk at any distance from its end which may be thought desirable, and holding it firmly forwards, the operation is easily completed by a pair of curved scissors. I am not aware that any instructions on this point have as yet been given, and having in one case experienced great difficulty in attempt to operate by the aid of sight, I think it well to mention it.

To these cases I may add a fourth, in which I have recently excised the eye for melanosis. In this instance we had also the phenomenon of detached retina as the earliest symptom. Afterwards, as is usual, the symptoms simulated chronic glaucoma, rather than the acute form which occurred in the preceding cases.

Case IV.—Mrs. H., æt. 40, was sent to me last March by Dr. Tidy, of Hackney. For a few months her left eye had been failing. She had experienced no pain. By ophthalmoscopic examination I found that the retina just above the outer side of the disc was detached, and that a peculiarly black appearance existed behind it. The media were quite clear, and nothing could be seen by the unaided eye. I wrote to Dr. Tidy, suggesting the probable existence of melanosis, but I did not feel sufficiently confident in my diagnosis to urge an immediate operation, and our patient could still see fairly in certain directions, and was very unwilling to believe her ailment a serious one. On November 27th she again called on me. The eye was now quite blind. The pupil was dilated and fixed, the tension of the eye considerably increased. By the unaided eye I could easily perceive the reflex

of some growth at the bottom of the eye. A yellow, almost metallic, appearance was produced. With the ophthalmoscope I could not make out anything more definite than that the retina was very extensively detached. Latterly Mrs. H. had had frequent attacks of pain in the globe, but of no great severity. I now urged her to submit to an immediate operation, and we removed the eyeball the same afternoon. The diagnosis was confirmed by our finding a large mass of melanosis in the substance of the outer part of the choroid, by which the retina and part of the choroid had been pushed inwards. The mass was as large as a cherry, and of sepia blackness. It showed under the microscope numerous cells containing nuclei but no pigment, and many other much larger pigment cells. In addition to the large bodies which were loaded with pigment, the latter also existed as free granules in great abundance. The growth had not perforated the sclerotic at any part. The lens and what remained of the vitreous body were clear.

Mrs. H. recovered well from the operation, and is at present in good health. It is now two months since the excision.

TWO CLINICAL LECTURES ON CASES OF INFLAMMATION OF THE OPTIC NERVES.*

By Jonathan Hutchinson, F.R.C.S.

Lecture I. (*October 24th.*)

The term *neuritis* had long been in use amongst pathologists before the introduction of the ophthalmoscope. It implied a belief that in some cases nerve trunks themselves inflame, and although the term was often employed without any demonstration of its applicability, yet there can be no doubt that it was often quite correctly used. The ophthalmoscope has enabled us to see easily, through transparent media, and under considerable magnifying power, the termination of one of the most important nerves in the body, and we are now perfectly familiar with all the various phenomena which attend the different stages of its inflammation. It is true that we see only the end of the optic nerve, but various reasons, especially the facts supplied by *post-mortem* examination, lead us to believe that in most cases what we see at the optic disc is a fair indication of the processes which are also going on in other parts of the nerve trunk.

Here I must ask beginners in ophthalmoscopic studies to draw a very definite distinction between the retina and the optic nerve. The retina, we all know, is not, as is popularly supposed, the mere expansion of the optic nerve. It is a highly complex structure, of which the optic nerve tubes constitute but a very small part. Of this fact we see abundant illustration in pathology. The nerve may be diseased without any material implication of the retina, and the retina may inflame without the nerve. It is true that in many cases both suffer together, but even when

* These lectures were delivered at Moorfields on the evenings of October 24th and October 31st. They were addressed to the students attending the Ophthalmoscopic Demonstrations.

they do so almost always one is implicated out of all proportion to the other, and nothing is more common than for us to see cases which we can definitely call *neuritis* or *retinitis* respectively, the inflammatory process being almost wholly restricted to one or the other structure.

The subject upon which we now propose to concentrate our attention is *neuritis*. I shall select as examples of it cases in which the retina was almost wholly exempt from implication. The group of neuritis cases, as far as the optic nerve is concerned, comprises almost all those in which blindness, partial or complete, follows severe "head symptoms." If a patient has suffered for some time from severe pain in the head, giddiness and vomiting, and amaurosis has followed, you may conjecture very confidently that his optic nerves have been inflamed, and that the loss of sight is caused directly by their disorganization. This group of symptoms rarely or never attends cases of retinitis or any other disease of the eye itself, and it is found in connection with neuritis, because this latter condition is almost always produced by disease within the cranium. In further proof of this statement, we may just remark that not unusually paralysis of other nerves, often in the form of hemiplegia, and very frequently indeed in that of loss of smell, attends this kind of amaurosis. By some the term "descending neuritis" has been applied to the disease under consideration, with the intention, I believe, of implying that the inflammation usually travels from within the cranium to the eye—as it were, *descends* from the central organ.

The phenomena of optic neuritis, as seen by the ophthalmoscope, differ very much in different stages, and the recognition of the clinical fact that the stage of acute inflammation with effusion is always transitory, and often very short indeed, is most important. Unless we allow for this, and unless we learn how to recognize the results of past neuritis, as well as the evidences of its actual presence, we shall be constantly making mistakes.

Before describing these phenomena we must call to mind the anatomy of the nerve with which we have to deal —that it consists, like other nerve trunks, of a bundle of nerve tubes, but differs from almost all others in the abundance of the neurilemma by which these tubes are invested and protected, and in the possession of an additional protection in the form of a strong fibrous sheath external to the neurilemma, and easily separable from it. The remarkable strength of the neurilemma is observable from the chiasma downwards to the eye. The external fibrous sheath is derived from the dura-mater, and is only present from the optic foramen to the eye. If we follow the nerve still further backwards from the chiasma to the corpora geniculata, we shall find it soft and with comparatively little neurilemna, but in large part having an investment of pia-mater. Now, when neuritis occurs it is doubtless the neurilemna which is directly involved. It is the nuclei of this structure which proliferate, and by which the new cells are produced. It is into the meshes of this tissue that the effusions, solid or fluid, of inflammation take place. It is owing to the continuity of this structure that the inflammatory process travels from one end of the nerve to the other. Bound down by this structure, and also by the external fibrous sheath, the tubes themselves must of necessity be compressed most injuriously by the accumulation of new cell elements and fluid effusions between them. Not only will the nerve tubes be compressed within their unyielding sheath, and thus have their function suspended, but at the same time we shall also see compression of the little artery and vein, which pass in the nerve substance for the supply of the retina. By *à priori* speculation we should expect neuritis of the optic nerves to produce, 1st, blindness, owing to compression of nerve tubules; 2nd, diminution of the central artery of the retina, owing to compression of its trunk; 3rd, turgidity of the central vein of the retina, owing to compression of its trunk; 4th, visible evidences of inflammatory effusion at the

exposed end of the nerve. Amongst the symptoms which might, with less confidence, be expected, are—5th, various subjective evidences of disturbed function, such as flashes of light, &c.; 6th, pain and headache, from the swelling and tension of a structure which cannot yield (such pain, being either near the part implicated, or referred to some other); 7th, evidences of reflex or sympathetic disturbance, such as vomiting.

If we may anticipate a little, I will here state that in practice we almost always find neuritis of the optic nerves symmetrical. If it occurs in one eye before the other, it almost always quickly shows itself in the one which at first escaped. This fact suggests that the inflammation starts from above the chiasma, or exactly at that part, and thus finds its way downwards into both nerve trunks. We may further state as the result of the clinical observations of authors that optic neuritis may occur in connexion with the following causes:—1st, tumours in the brain; 2nd, arachnitis, especially when at the base of the brain; 3rd, fevers, with head symptoms (probably arachnitis); 4th, tubercular meningitis. Dr. Hughlings Jackson, to whom we are indebted for most valuable facts and reasonings on this subject,* holds that disease of almost any part of the cerebrum or cerebellum may be productive of optic neuritis; and, secondly, he has made the very important observation that when in these cases hemiplegia occurs, as it often does, it is usually on the left side of the body.

I have already said that the appearances by which we recognize neuritis in its acute stage are often transitory. Only now and then do we see good examples of it, whilst instances of its results are seen very frequently indeed. During the present course I have shown you not a few

* *Vide* Ophthalmic Hospital Reports, vol. iv, page 389, "Observations on Defects of Sight in Diseases of the Nervous System." By J. Hughlings Jackson, M.D., Assistant-Physician to the National Hospital for Epilepsy and Paralysis.

instances of atrophy, the consequence of neuritis, but no opportunity has yet occurred for demonstrating the phenomena of the early or aggressive stage. Should the patient come under our observation when the neuritis is just beginning, we may find probably only one part of the optic disc affected. At that part the disc is elevated to a degree very easily appreciated by the binocular.* The margin of the choroid is no longer to be distinguished, being concealed by the exudation in the nerve in front of it. The nerve itself, instead of showing its normal pink tint and smooth surface, is of a lilac, or light grey, colour, and looks fluffy, much as if cotton-wool had been carded until all its fibrils radiated outwards from a centre. From this peculiarity I have been in the habit of calling it "the woolly optic disc," and others have, I believe, found the term very fairly descriptive. This radiating appearance has arrested the attention of all observers, and is very well represented in Liebreich's Atlas, fol. xi, figs. 6, 8, and 9, the letter-press to which contains a concise but very graphic description of the changes.

We have supposed that we were fortunate enough to see the case at the very earliest period, and before the whole of the disc had become involved. A little later and we shall find the edges of the disc wholly concealed, the disc appearing very much larger than natural, owing to the extension of the exudation over the choroidal margin. We shall notice also that the trunks of the vessels passing to the centre of the disc can no longer be traced, but are buried in the new material, that the branches of the arteria centralis are much smaller than usual, and those of the veins much distended. It is this stasis of the capillary circulation amongst the inflammatory materials which gives to the latter its light lilac tinting. Sometimes extravasations of blood of greater or less size occur, owing

* So real and considerable is the elevation by swelling in some cases, that the erect image of it may be seen in a myopic eye without the use of concave lens. *Vide* Liebreich, page 33.

to the venous distension, but these are exceptional in cases of pure neuritis, and are much more common in certain other forms of disease.

The recognition of neuritis in its early stages is easy, for the changes are very marked. At this stage the diagnosis chiefly concerns neuritis from intra-cranial causes (that is pure neuritis or descending neuritis), and those other forms of neuritis complicated by retinitis, and produced usually by blood disease, such as syphilis and albumenuria. On this point the limitation of the changes to the immediate neighbourhood of the disc itself is one of our principal guiding facts. In syphilitic retino-neuritis the whole retina is usually affected, and the neuritis element is small.

The first case which I shall mention is one of peculiar interest, from the fact that I have had the patient under observation from the very commencement of the disease. It is that of James Oakley, a man aged 35, whose eyes you have all examined this evening. He is at present a stout, healthy-looking man but he speaks a little thickly, and is excitable in his manner. He is quite blind as regards the left eye, but can see large objects with the right. The left pupil acts a little when exposed to light. The pupil of the right is quite fixed. In a dull light the two pupils are of equal size. In a bright one the left is considerably smaller. I ask your especial attention to this fact, for you will note that it is the iris of his blind eye which acts, whilst that of the eye he still sees with is motionless: a fact in further proof of what we have often adverted to, that the activity of the pupil is not by any means always in direct ratio with the perception of light. With the ophthalmoscope we find very similar conditions in the two eyes. The media in both are quite clear, and in both the optic discs are dirty white and atrophied. Their whiteness is not of the clean milk white of mere anæmia;—of what we call *par excellence* "white atrophy." It is rather of a dirty bluish grey. The discs are somewhat depressed in the

middle; their margins are tolerably abrupt, but here and there a little jagged, with a few minute dots of pigment. The branches of the central vein are of a fair size, but those of the artery are very small. The retina adjacent to the optic disc is perfectly transparent, and the vessels can be easily traced. The choroid is perfectly healthy. Altogether there is but little in the present condition of the discs, which would enable even an experienced observer to say whether or not neuritis had preceded the atrophic changes now existing. Yet, as we shall see presently, it is quite certain that it did so. I ought to have stated that Oakley is at present partially paralytic in his right arm and leg; that he is exceedingly susceptible to the influence of stimulants, and that his wife reports that he is subject to short fits of uncontrollable passion. Before his illness he was a man of remarkably quiet temper. His hearing, his smell, and his taste are fairly good; he has a large appetite, and a craving for stimulants.

Having thus described in detail his present condition, let us now turn to the very interesting facts of his illness. Up to October, 1864, he was in the enjoyment of good health, and was engaged in one of the large city houses. On October 20th (Thursday), he came to me with the statement that he had suddenly become blind in his left eye. On rising from bed on the previous Monday, he had found his sight dim, and on trial he had found that it was the left eye only. During the next two days the eye got rapidly worse, and he could now only just perceive large objects with it. On the morning of his application, for the first time, his right eye also failed, and for a few minutes he was almost blind. Both pupils were large and exceedingly sluggish. In the *left eye* the margins of the optic disc were ill-defined, being covered by a halo of greyish white. The veins were exceedingly turgid, the arteries very small. Slight pressure on the globe sufficed to empty the veins entirely on the surface of the disc. The media were quite clear, and excepting at the optic disc, the fundus was quite

normal. The *right eye* showed conditions exactly similar, but less advanced. The arteria centralis was larger, and the vein not so turgid. In both eyes the surface of the disc had the appearance of being swollen and elevated. We had, therefore, a very marked example of optic neuritis in its earliest stage. Let us note the constitutional symptoms which attended it.

They were exceedingly slight. During the last week our patient had had frontal headache, "like something shooting above the eyes," but it had not kept him awake; he had on the contrary been very sleepy. Until this illness he had scarcely known what it was to have a headache. During one day previous to his admission he had been very giddy, and this also was a symptom which he had never experienced before. He had had no vomiting. He looked pale, and his lips were rather dusky, and on being closely questioned, said that he had lost flesh of late, had suffered very frequently from bad colds in the head, and had been accustomed to perspire very freely. Beyond this we could make out nothing as to symptoms of disease of the brain. The remedies prescribed consisted in hot pediluvia, purgatives, and a blister to the back of the neck. On October 24th he was quite blind with the right eye. The discs in both eyes were very much more swollen, the edge of the choroid was quite concealed. There were no extravasations. He reported his headache as better, but he had still some severe pain. On the 27th he was in total darkness. The ophthalmoscope, as before, showed the discs swollen, elevated, and woolly. The turgid veins were seen dipping into the effused lymph, but no branches of the artery could be distinguished. We now ordered him a grain of calomel every two hours, and another blister at the nape of the neck. The effect of the mercury was apparently very decided. It was pushed to salivation, and ten days after its commencement the quantity of lymph was very much less in both eyes, and with the right eye he could count fingers. From this time the absorption of the lymph was steady,

but beyond the perception of large objects, he did not materially improve in sight.

After this, however, the cerebral symptoms developed themselves. He became hemiplegic on the right side, and to a large extent lost the use of language. He was now admitted under my care into the London Hospital, where he remained from December 22nd to March 7th. During the whole of this time he was very ill, paralytic, helpless, and childish. His paralytic symptoms were complicated, and difficult of explanation. The following notes written at his bedside on January 3rd and 4th, describe his condition in some detail. It will be seen that at this date we had the curious symptom, which is still present, of dilatation and fixity of pupil in the better eye; (probably owing to disease both of third nerve and of the vasomotor fibres on this side.)

"*Tuesday*, *January 3rd*.—Tongue very dry. Lips dusky. He is conscious, told me my name, told me that he had pain in his stomach, although articulating with great difficulty. When asked where he was he only mumbled and did not produce any intelligible word. His left eyelid drooped so as almost to cover the eye. His face was drawn to the left side, but not very much. On testing the sensation in his cheeks, he gave us to understand quite clearly that he could feel much better on the left side. On touching his eyes he winked strongly with the left eyelids, but could not close the right ones. Both pupils were widely dilated, and there was no material difference between them.

"I found his right foot very much hotter than his left. His right was very hot, his left cool. Both had been equally well covered.

"I could not detect any difference in the relative temperature of his two upper extremities. He gave me to understand that he had no headache. His bowels had only acted twice in the week; each time after castor oil. His water passes away unconsciously, but he is able at

times to let the nurses understand. He can draw his right leg up, but he moves it very little."

"*Wednesday, January 4th.*—Both his feet to-day are equally cold and pale, decidedly chilly. There is no decided difference in the temperature of the two arms. In other respects he seems much as he was yesterday."

A few days ago he was restless, throwing the clothes off, &c., but during the last three days he has lain in a quiet sleepy condition. When roused, his intellect seems clear. I pinched his two hands, and asked him which he felt best in, and he said the left, and I found that he clearly understood what he meant. He can move the right arm a little, but only very little, and when told to close his eyes the muscles on the right side do act a little, but very much less than those on the left. He can open his right eye easily, and let it drop shut. The left eye he cannot open more than a very little.

"There is no very positive strabismus in any direction, but he moves his left eye rather more slowly than the other. Both pupils are dilated, but the right is more than twice the size of the other. Both are absolutely motionless. In both eyes the optic disc is pale and grey, and the veins and arteries are very much diminished in size.

"On the right side he can hear a watch pressed over his ear, but he loses it at the distance of one inch. On the other side he appears to hear well.

"I do not perceive any difference in the relative temperature of his two ears. The masseter and temporal muscles on the right side act very much less than those on the left and appear to be wasted.

"He has never been sick, takes his food well, and eats mutton chops. He says he eats on his left side. He sometimes splutters in drinking, but, as a rule, he swallows well. He has never had any convulsions."

The following is the note on March 14th. "Oakley left the hospital a week ago. He had been improving for a fortnight before his discharge, both in general health and

in intelligence. He was still quite paralysed in his right limbs, and the right side of his tongue also looked thin and flabby. His tongue did not materially diverge. The ptosis of his left eye had nearly passed off. He could speak clearly, and seldom used words wrongly. Had great difficulty in getting him to remember where he was; indeed, he never told me right. Sometimes he would reply, "In St. Mary Axe," sometimes "In Austin Friars," sometimes at the "Insurance Office;" and, although I always explained to him where he was, he could not retain the knowledge for more than a few minutes. He never forgot my name and always recognised me. He was very cheerful, and usually answered questions quickly. At the ends of sentences he would mumble and become unintelligible. This evening his wife calls and reports that since his return home he has still further improved. She even thinks that he can see at times with the right eye. He is dressed each day, and with a little assistance can stand, although still paralysed in the right limbs. He eats well. She says that his memory is very fair, and that he can say almost anything, only he often miscalls things."

On May 8th.—The notes state that he could see large objects, and that although his face was still a little to the left, he could whistle and could frown on both sides of his face. "Speech and use of language perfect, excepting a stutter in commencing sentences. His right arm he cannot move in the least, and sensation in the right limbs is very much impaired. He feels a touch very indistinctly, and cannot appreciate the two points of the compasses when three inches apart. His lower extremity is not so completely paralysed. He sleeps well; has no headache; his smell seems tolerably good."

From the date of the last note up to the present time he has continued steadily to improve until reaching the condition in which you have seen him this evening, and which I have already described.

If we attempt to speculate on the nature of the intra-

cranial disease with which the neuritis has been associated we shall undertake a very difficult task. The remarkable manner in which the man has got rid of paralytic symptoms of the most severe kind, has lost his headache, and has regained good general health, seems to put out of view the existence of a cerebral tumour, and favours the hypothesis of arachnitis.

Had he permanent loss of smell this hypothesis would be yet further strengthened. I will now read the particulars of the case of another patient whom in former sessions you frequently had opportunities of examining, and in which we ultimately had a post-mortem examination.

Walter Snell, æt. 28, came to us at the Ophthalmic Hospital in a state of almost complete blindness from the effects of neuritis. He had just been discharged from the army in consequence of his condition. His optic discs were bluish-white, and somewhat depressed. The margins of the choroidal rim in the right eye were a little jagged, but in the left were well defined. The branches of the arteria centralis were much diminished, but still easily traced. Those of the vena centralis were of normal size. He appeared to be in good health and had no other paralytic symptoms. He complained much of pain in the right temple, holding his hand to the part. Whilst we were examining him he became faint.

The history of his attack was as follows :—Until a few days previous to January 1st, 1864, his sight was perfect; then his right eye began to fail, and on New Year's morning he went into the military hospital with this symptom. He had some pain about the right eye, and a blister was applied to the right temple, which relieved it. Five days after his admission his left eye in the course of one night became quite blind. At this time he had very severe pain in the forehead over each eye. The pain was so intense and so unremitting, that he thinks he never slept for one week. His head was shaved, and a blister ap-

plied across the forehead. This blister gave him the greatest relief, and he was without pain for several weeks afterwards. He left the hospital after a stay of three months and came up to London, where he was admitted under my care on April 28th. At this time, as I have already stated, all evidence of recent neuritis had passed away. He was quite blind of the left eye, and could only just perceive large objects with the right.

In our inquiries as to the man's previous history, we learnt that about four years ago he had received a violent blow on the left forehead with a knobbed stick. He was stunned by the blow and was under treatment for several days after it. On careful examination of the part said to have been struck we could not detect any evidence of thickening, and during the three years' interval between the injury and the neuritis he had enjoyed excellent health. He had never had syphilis. He had never been liable to giddiness or to headache; nor had he had any vomiting whatever. He had served in the Crimea and in Bengal, and in the latter place had suffered from ague.

About a fortnight after Snell commenced his attendance at the Ophthalmic, I was one day summoned hastily to his brother's house, with the statement that he had had a fit. I found him incoherent in his talk, and slightly, but decidedly, weak in the left limbs. During the next few days the paralysis diminished.

On June 20th, we admitted Snell into the London Hospital in order to be able to watch the treatment more continuously. Mercury was administered to salivation, and with some apparent benefit, but it was not permanent. He remained in the hospital four months, and during almost the whole of this period suffered from intense pain in the head. Often for days together he would lie in a semi-stupified condition, apparently the result of the severe pain. His pupils were usually widely dilated and fixed. He took large doses of morphia with very little result. Hypodermic injections were used, and on two occasions he

was put so fully under the influence of opium that his pupils were contracted to mere points.

He would frequently pass his urine and fœces unconsciously.

At length, without apparent cause, he began to improve, and in November he was able to leave the hospital. From November to February he attended at the Ophthalmic. He was now quite blind, but had almost wholly recovered from the weakness of his right limbs. He still had severe pain in the head. Vomiting now frequently occurred. He had complete loss of smell and taste, so that when he would ask for ale, and water was given him, he did not seem to know the difference. In January and February he was confined to bed at home, and had a succession of epileptic fits, and became at times almost maniacal. After a succession of convulsions he became much exhausted, and died February 20th.

At the autopsy we found no evidence of injury to the skull, either external or internal. The general mass of the brain was remarkably soft. Beneath the anterior lobes and extending backwards in the middle line almost to the crura, was a large quantity of organised inflammatory material. The olfactory bulbs, the optic nerves and adjacent parts were matted together by very firm adhesions, and the cerebral lobes broke down in the attempt to remove them. The optic nerves were so much softened that it was difficult to distinguish them from the lymph in which they were imbedded. Three small isolated collections of thick semi-purulent fluid were found in the midst of the organised lymph. There was nothing that looked like tubercle or any form of new growth. The membranes on the upper surface of the hemispheres were healthy, and, excepting the conditions described, no disease was found within the cranium.

The disease appears in this instance to have been essentially a local chronic arachnitis, but its cause is still very obscure. As an interval of three years of health had

intervened between the blow and the cerebral symptoms, we can scarcely suspect any connection between them; otherwise the case which I am about to relate might have led us to entertain such a supposition.

Three weeks ago I was called to Epsom, in consultation with Mr. Owen, of Leatherhead, to see a little boy who was suffering from cerebral symptoms, supposed to be consequent on a fall down stairs, in which his head had been struck. Our patient, when I saw him, was almost wholly insensible. His left limbs were weak, and his right eye had a dilated pupil, was insensible to light, and the globe itself remained motionless in the orbit. It appeared that all the ocular muscles were paralysed. He moved the other eye freely and its pupil was fairly active. The conjunctiva of the right eye was congested and in a state of chemosis. There was a turgid vein on the right temple, which had no fellow on the left. From these conditions I diagnosed arachnitis at the base of the brain on the right side; judging that the right cavernous sinus was probably obstructed by thrombus, and that the adjacent nerve trunks were paralysed by the effusion of lymph about them, I had used the ophthalmoscope and had found evidences of neuritis at the optic disc. The injury to the head had been received three weeks before any symptoms of cerebral disease had set in, and had been almost forgotten, but it was now remembered that the boy had throughout that interval seemed heavy and stupid. His first serious symptoms had been a shivering fit, and after that, sickness and constipation. He died a few days after my visit. A post-mortem examination was obtained by Mr. Owen, who informed me that our diagnosis had been fully confirmed, and that the nerves passing into the right orbit had been found matted together by recent lymph.

Lecture II., October 31st.

Amongst the patients whom we have examined this evening, was one in whom we conjecture that the neuritis may possibly have been of syphilitic origin. At any rate, she had had constitutional syphilis some years before her attack of neuritis, and there is an absence of any indication of other cerebral disease, such as tumour. If syphilis is a cause of optic neuritis it is probably not very frequently so. I have seen, however, a few patients, especially the subjects of inherited syphilis, in whom amaurosis with atrophy of the nerves had occurred. We know that deafness, probably from nerve disease, is fairly common in inherited syphilis, and occurs occasionally as a consequence of the acquired form. We know also that paralysis of the fifth nerve, or of any of the motor nerves, of the eyeball is frequently met with as a syphilitic disease. Whenever syphilitic disease of a nerve trunk occurs it is always as a tertiary symptom, and at a long interval after the primary infection. Retinitis, as you are aware, is on the contrary, a secondary symptom, and usually occurs co-temporaneously with iritis three to six months after the primary disease.

Mrs. A., aged 30, is now a healthy-looking woman, she has attended at our demonstration this evening, and you have all had opportunities for examining her optic discs. Both her pupils are widely dilated and quite fixed. She has the barest perception of light, and cannot tell even where the windows are. Both her optic discs are of grey-white colour, quite flat, their margins are somewhat jagged and indistinct, but not much so. At one or two places the trunks of the vessels close to the margin of the optic disc are obscured by a white film, but this is very limited. In each eye the central artery and vein are of fair size.

Mrs. A.'s history is that up to last September she could

see perfectly, but for years she had been subject to pain in the occiput, giddiness, and noises in the ears. She has been married ten years, and five years ago her husband gave her the venereal disease. The local symptoms were followed within a few weeks by a rash and sore throat. Since that she has been much under medical treatment, and has had five miscarriages and one living child. This child in its infancy had specific symptoms. Her amaurosis commenced in September last. Loss of appetite and sickness were the first symptoms. She had afterwards great pain in the head, and was for some weeks confined to her bed and almost insensible. At one time her left limbs were weaker than the others, but they were never paralysed. On recovery from her illness she found that she was quite blind and had utterly lost her smell. Her hearing remained perfect. Eight months have now elapsed since her recovery, and during this time she has been free from symptoms. She at present appears in good health, is free from headache, has no sickness, and, excepting the complete amaurosis and anæmia, she has no symptoms referable to the nervous system.

A second case, with almost a precisely similar history, was before us a few weeks ago. The subject of it is a married woman, æt. 35, who suffered from primary syphilis followed by rash and sore throat ten years ago. She thinks that she took mercury for it, but her gums were not made sore. Ever since she has been liable at times to scattered blotches on the skin and sores on the tongue. She does not appear to have ever had periostitis of superficial bones. Her eyes began to fail about five years and a half ago, and within three or four months of the first perception of fog she had become totally blind. She had at the time a little headache, but there do not appear to have been any concomitant symptoms. She complains now of great depression of spirits, but, excepting the amaurosis, she has no paralysis. Her smell is good, as also is her hearing and

sense of touch. With the ophthalmoscope precisely similar conditions were found in the two eyes.

The media are perfectly clear. The optic discs are very conspicuous and very white, round, abruptly margined, quite destitute of pink stippling. They are somewhat depressed in the centre by atrophic shrinking. The arteria and vena centralis are each of almost normal size, and being placed on a white ground are very conspicuous. The choroid is healthy. It must be observed that in this case we have no proof that any stage of neuritis ever occurred. The conditions now present are suggestive rather of white atrophy than of the results of inflammation. In the history of the original attack we also miss, as regards the symptoms of pain in the head, vomiting, &c., which usually attend neuritis. Its probable connection with syphilis, however, leads me to mention it here in connection with the preceding case.

[To be concluded.]